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A New Medico-Legal Journal

The decision to launch a new scientific publication is not made lightly, and we have embarked on this venture for several very cogent reasons.

Although in one or two countries there exists a small number of journals for forensic publication, the facilities offered are very circumscribed and restricted. There is a considerable contemporary need for a journal (with international scope) which can devote itself exclusively to the many readers and research workers in this important branch of knowledge and practice.

Diverse and apparently unrelated fields of scientific inquiry will here find a rational and neighbourly place, e.g. anthropometry and serology, medicine and toxicology, ballistics and traumatic pathology, the physics of stress and strain and head injuries, cardio-respiratory physiology and the objective detection of lying, psychiatric developments and legal concepts of responsibility, etc.

At present important forensic contributions must necessarily be scattered widely for publication. This undesirable dispersal of papers (sometimes in obscure or not easily available journals) interferes with their ready appraisal by those who can appreciate their medico-legal significance.

The intensive research developments taking place in a multiplicity of laboratories and institutions have accentuated the difficulties facing all authors seeking publication of their papers, but this is markedly enhanced in the case of forensic practitioners by the poverty of media for the publication of case reports, reviews, original observations, etc. It is the more so in the case of medical practitioners in this field as they find themselves increasingly in competition for space with contributors to journals devoted primarily to clinical medicine or pathology.

Moreover, it is desirable for the average reader to find the great diversity of the material which makes up his professional field of interest in the more convenient compass which one journal such as this can provide efficiently and inexpensively.

The increased status and the prestige of the subject of Forensic Medicine have received professorial recognition in recent years in universities all over the world. It is equally fitting to endorse this recognition by the establishment of an international journal solely concerned with this subject. We hope that it will, in its turn, do much to stimulate further forensic research and writing.

Blood Groups and Skin Colour

Their Genetics in Human Anthropology *

M. Shapiro, M.B., B.Ch.
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The function of the geneticist (and of the physical anthropologist) in his approach to the subject of racial differentiation is to present objectively and scientifically the innate characteristics which distinguish different human stocks. Such a presentation, therefore, excludes consideration of cultural patterns or moral codes; it also excludes all questions of 'racial superiority' except in so far as it may be possible to produce evidence of hereditarily determined qualities whereby one stock may be expected to possess an advantage over another in adapting itself to a particular environment.

All physical attributes are the result of environment, of heredity, or both. Skeletal structure and skin colour are 2 physical characteristics which, more than any other, have influenced the popular conception of race throughout the ages. But we are all familiar with the fact that skeletal structure of a particular human stock may be modified greatly by such external influences as climate and nutrition. Thus, the average height of the present generation of white South Africans is substantially greater than that of their English, French and Dutch ancestors of only a few generations ago. Skin colour also appears to be too strongly influenced by environmental factors to form a solid basis for racial differentiation. Without exception the indigenous races who occupy the tropical and sub-tropical regions of the world are all deeply pigmented. The Oceanic Negro of Papua with his woolly hair and black skin shows a strong physical resemblance to the African Negro, yet genetically he is probably as far removed from the African as from the blonde Caucasian.

For more fundamental evidence of racial differentiation we should, therefore, prefer criteria which are minimally affected by environment, which are not subject to mutation at any very rapid rate, and which are determined by known genetic mechanisms. The blood group antigens fulfil all these requirements. They also have the advantage that they are entirely uninfluenced by conscious selection, either on the basis of personal preference (as in choosing a mate) or of popular prejudice which, since time immemorial, has distinguished certain ethnic groups and even sects as superior or inferior to others. As Boyd[1] has so aptly remarked:

> 'In certain parts of the world an individual will be considered "inferior" if he has, for instance, a dark skin, but in no part of the world does the possession of a blood group A gene or even an Rh-negative gene exclude him from the best society.'

However, a racial classification based on the blood groups differs in one fundamental respect from one based on physical characteristics such as skin colour, hair texture and skeletal structure. It is quite impossible to

*The substance of this paper was contributed to a *Symposium on Race* at a meeting of the Medico-Legal Society (Johannesburg) in November 1952.

determine the race of an individual from his blood groups. At present we can define at least 9 separate blood group systems comprising 25 blood group factors, each of which can be detected accurately with appropriate testing sera. With these reagents, more than a million different human types can be distinguished. Like his finger-prints, the blood group complex of the individual is immutable from birth to death. But one can no more tell the race of any person from his blood groups than from his finger-prints; a pygmy of the Efe forest may have an assortment of blood groups identical with that of a Maori or an Englishman. It is only when we compare the frequencies with which the individual blood factors occur in, say, 500 pygmies and 500 Maoris, that marked differences between the one group and the other become apparent. Compared in this way, we find that the indigenous populations of the world can be classified broadly into 6 main groups[5]—the Early European, the Modern European or Caucasian, the African, the Asiatic or Mongolian, the American Indian and the Australian aborigine.

This classification corresponds well with the geographical distribution of the human species on the face of the earth.

The Blood Groups

In Fig. 1 are shown the gene frequency distributions of 2 blood group systems—A B O and Rh—as they occur in 22 ethnic groups. These have been calculated from the reported observations of numerous investigators and are here represented graphically to facilitate comparison (*vide* References).

1. The members of the *Early European group* (of which the Basques of Spain are the most typical example extant) have the highest frequency of *r* (the Rh-negative gene). They are almost devoid of *B*, and A_2 is relatively infrequent. They probably owe their characteristic serological pattern to the fact that they have been influenced less by Mongoloid admixture than have most of the other peoples of Europe.

2. The members of the *Modern European group* all have fairly high frequencies of *r*, and A_2 is present in relatively high proportions. The *B* gene is variable in frequency. It is highest in India and diminishes in all directions from that peninsula. According to Candela[6], the B factor was first introduced into Europe by the Tartars and the Huns about the fifth century A.D. and its importation was continued by successive Mongolian invasions during the following 10 centuries. The greatest influence was probably effected by the hordes of Genghis Khan which over-ran the greater part of Europe and the Near and Middle East at the end of the 12th century. The Egyptians, Arabs and Moslem Indians display all the serological features of the Modern Europeans, but their gene frequencies indicate that they have been influenced by Mongoloid strains to a greater extent than have the peoples further West. Also, substantial admixture of Negroid strains is apparent from their genetic patterns.

3. In Africa, below the Sahara, is the *Negroid group*, markedly differentiated from all the other peoples of the world by having a tremendously high frequency of R^0. The intermediate genes $\mathfrak{R}^0$ and $\mathfrak{R}^1$ (which are very rare in most other races) are also fairly frequent in Negroids. *B* occurs with about the same frequency as in Whites; A_1 is less common, but A_2 and the weaker subgroups of *A* are much more frequent. Linguistic and historical evidence suggest that the Bantu originated in the region of the sources of the Nile. The South African Bantu are probably fairly recent immigrants, having arrived in this country not long before the colonization of the Cape by White settlers. The frequency of the R^0 gene in Africa diminishes from South to North. In the Belgian Congo,[30] for example, it is already substantially lower than in the Transvaal. This dilution in the frequency of the R^0 gene is undoubtedly due to Hamitic, Caucasian and perhaps also Indonesian admixture which the South African Bantu largely escaped by timely migration to the southern extremity of the continent.

4. The members of the *Mongoloid group* all have extremely high frequencies of R^1, little if any *r* or A_2. It is significant that the gene R^1 dominates the serological patterns of all the peoples of the world with the exception of the Negroids, a fact which testifies to the extraordinary migrations by sea and over-land which have proceeded from Asia since prehistoric times.

5, 6. Two other groups (the *American Indians* and the *Australian Aborigines*) have been defined tentatively. Although their serological patterns are clearly predominantly Mongoloid, they do have certain distinctive features: *B*, A_2 and *r* are absent; A_1 and R_2 are more frequent than in other races. The genetic pattern of the Polynesians, of which the Maoris are an example, corresponds closely to that of the American Indians, a fact which lends support to the theory of migration which prompted Heyerdahl and his companions to undertake their epic voyage by raft from Peru to the Pacific Islands (the Kon-Tiki expedition).[17]

Differences in the frequencies of the genes for the MNS system and for several of the more recently discovered blood factors such as Kell, Duffy and Lutheran, add to the distinctive features of the races as defined here. In Table 1 are detailed the frequencies of the blood types in 7 blood group systems in 3 racial groups. The data for the South African Bantu are from a recent investigation and will be reported in greater detail elsewhere.

Although, as has been said, it is quite impossible to identify the race of a particular individual from his blood group complex, a knowledge of the racial distribution of the various blood factors may have some forensic application. It is a simple mathematical calculation to compute the frequencies with which particular combinations of blood factors may be expected to occur in different racial groups. For example, the Duffy factor is found in about 100% of Chinese, 66% of Europeans and only 12% of South African Bantu. The type Rh_o, Duffy-negative occurs in only 0.7% of South African Whites, but is present in 60% of Bantu. Thus, if a pool

TABLE 1: COMPARISON OF THE BLOOD GROUP PHENOTYPES PER CENT IN 3 RACIAL GROUPS

System	*Types*	*Chinese* (*Miller et al*)[20]	*English* (*Various Authors*)	*South African Bantu* (*This Author*)
A—B—O	O	45·6	43·5[17]	50·2
	A_1	27·2	34·8	16·7
	A_2	0·0	9·9	11·5
	B	22·3	8·6	17·4
	A_1B	4·9	2·6	1·8
	A_2B	0 0	0 6	2·4
M—N—S	MS	3 9	20 8[25]	9·8
	Ms	35·0	7·5	23·9
	MNS	5·8	26·7	12·2
	MNs	37·9	22·7	36·6
	NS	1·0	7·2	3·4
	Ns	16·5	15·1	14·1
P	P+	27·2	74 0[26]	92 0
	P−	72·8	26 0	8·0
Rh	rh	1·0	15·1[24]	1·4
	Rho	1·0	2·1	69·1
	℞ho	0·0	·	3·0
	Rh_1rh	11·7	33·8	14·4
	$Rh_1{}^wrh$	·	1·0	0·3
	Rh_1Rh_1	53·4	17·4	0·3
	$Rh_1{}^wRh_1$	·	1·1	0·0
	Rh_2rh	8·7	11·7	6·9
	Rh_2Rh_2	0·0	2·3	0·0
	Rh_1Rh_2	24·2	13·2	1·2
	$Rh_1{}^wRh_2$	·	0·4	0·3
	℞$h_1$rh	0·0	·	2·2
	rh′	0·0	0·8	0·8
	rh″	0 0	0·9	0 0
Kell-Cellano	K+	0 0	7·1[1]	0 6
	kk	100 0	93 0	99·4
Duffy	Fy^a+	99 0	66·0[7]	11·8
	Fy^a−	1·0	34 0	88·2
Lutheran	Lu^a+	?	7·6[25]	5 4
	Lu^a−	?	92·4	94 6

of blood should be found which might have been shed by a White person or a Native, the chances are over 80 : 1 that it is from a Native if this combination is present. Similarly, the type Rh_1Rh_1 is present in over 18% of Whites, but I have found only 5 examples of this type in 1,844 Bantu bloods. Recently, I had the opportunity of demonstrating the potential value of these facts to Dr. J. Friedman, Senior District Surgeon of Johannesburg. From a large number of White and Bantu blood samples chosen at random, he selected 60, marking each sample only with a number. Utilizing the comparative frequencies of the genetic combinations, I found that in 13 the chances in favour of the sample being either White or Bantu were less than 10 : 1. In these I did not hazard a guess. Of the remaining 47 samples, I identified 45 correctly. Crimes of violence, not infrequently preceded by a bloody struggle, are not infrequent in this country. At present there

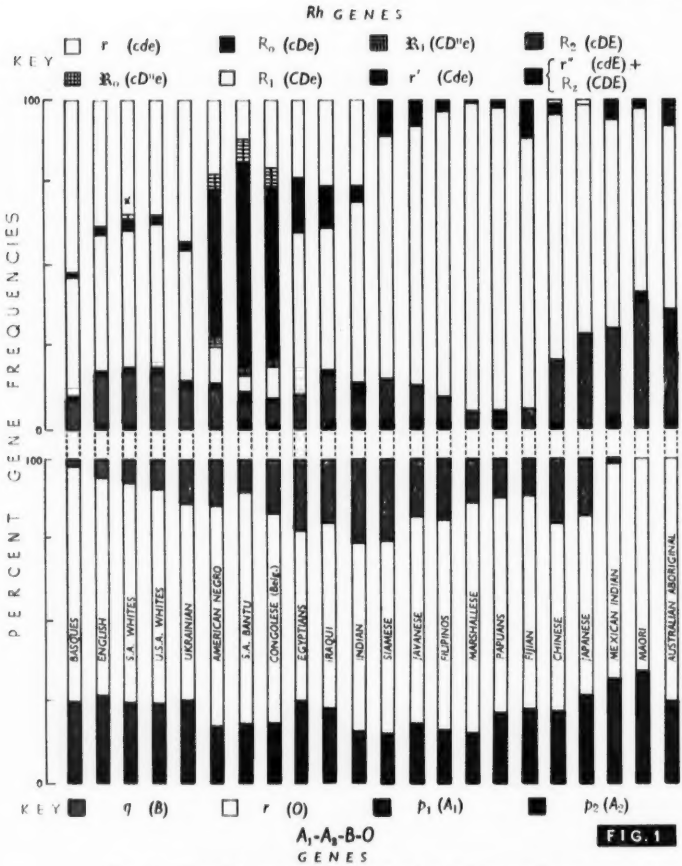

Fig. 1. Blood Group Gene Distribution in Various Races.

exists a legal prohibition against the involuntary procurement of blood samples from suspected persons. This militates very seriously against the use of serological methods for purposes of personal identification and it would appear, in the light of our present knowledge, that some modification in the law has become necessary.* Nevertheless, despite existing legal difficulties, the detailed blood grouping of wet blood stains found at the scene of a crime may, in certain circumstances, provide a valuable clue.

If we visualize the individual blood group genes as identifiable particles

* In the Union of South Africa in the *Criminal Procedure and Evidence Amendment Bill* (1950) a new section provides that the medical officer of any prison or gaol or any district surgeon 'may take or cause to be taken such steps, including . . . any blood test as he may deem necessary in order to ascertain whether the body of any such person bears any mark, characteristic or distinguishing feature or shows any condition or appearance . . .'

In certain circumstances magistrates may instruct that arrangements be made for a blood test.

These provisions have not yet been enacted.—*Editor.*

inherited according to Mendelian laws, it can easily be understood that miscegenation of 2 racial groups in, say, equal numbers, will result in a distribution in the first hybrid generation in proportions which will be the averages of the frequencies in the parental groups. If the hybrid group were thereafter to be insulated from further infusion of genes from either of the parental groups, the gene frequencies should remain practically constant throughout each succeeding generation. In Fig. 2 are shown the effects of miscegenation between Whites and Negroes on the Rh gene

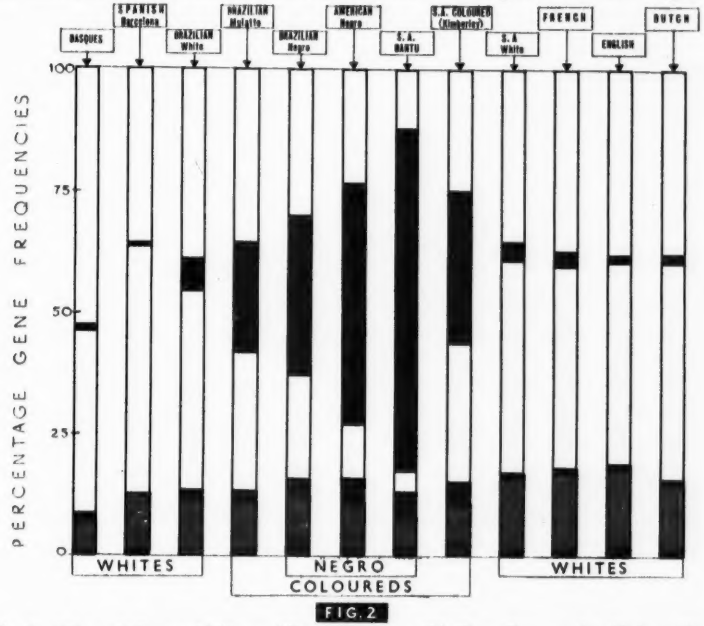

FIG. 2

Fig. 2. Effect of Caucasoid-Negro Miscegenation on Rh Gene Frequencies (Colour Key as in Fig. 1).

distribution as they have occurred in South Africa and Brazil. Fig. 3 demonstrates a method of estimating the percentage mixture in hybrid groups, such as Eurafrican and Cape Malay. Correlation of the results obtained from a sample of 450 Coloured persons (Kimberley, South Africa) investigated by me showed them to be a mixture of approximately 42% White and 58% Bantu. The deviation from 50% in the direction of preponderance of Bantu genes is statistically significant. It may be attributed to further hybridization having occurred more frequently between Coloureds and Bantu than between Coloureds and Whites.

Skin Colour

The genetics of skin colour are much less clearly understood. The specific factors concerned cannot be tested for with the same accurate techniques as are available to us in the case of the blood group antigens.

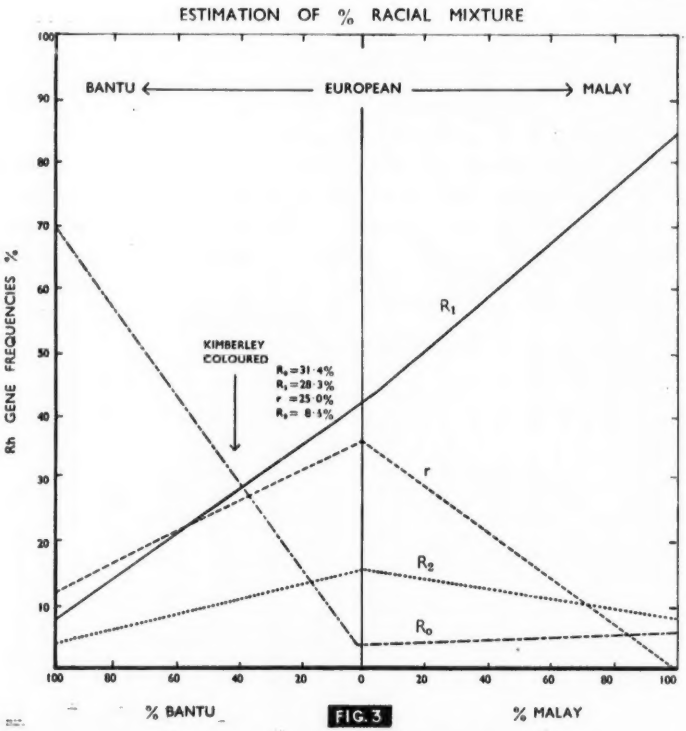

Fig. 3. Estimation of Percentage Racial Mixture.

Assuming, with Davenport,[10] that skin colour is determined by only 2 pairs of genes, the first filial generation of black and white parentage will all be intermediate in colour, each individual containing 2 'white' genes and 2 'black' genes. However, if 2 such hybrids mate, not all their progeny will possess 2 of each variety of gene. Statistically, one out of 16 will possess no 'white' genes at all and will be black; 4 will contain 3 'black' genes and one 'white' and these will be darker than either parent; 6 will be intermediate like their parents and will possess 2 of each variety of gene; 4 will contain only one 'black' gene and these will be lighter than either parent; and one will contain no 'black' genes and will be white. Human nature and popular prejudice being what they are, we may expect that the lighter coloured progeny will seek mates who are not darker skinned than themselves, or be forced to accept mates who are similarly or more deeply pigmented and so the tendency will be towards differentiation in 2 directions—either towards white or towards black; and, if we may allow our fancy to wander further, to visualize some future generation of segregated 'Coloured' persons, we may find among them a colony of 'White'

persons indistinguishable in skin colour from their White ancestors. But if we examine their blood groups we will find, as we find to-day, that they have a genetic pattern corresponding to what would be obtained from a mixture of White and Negroid strains.

Davenport's theory is undoubtedly an over-simplication of the genetics of skin colour since the gradations which may occur are more numerous than postulated by him. More than 2 pairs of genes are probably involved. In addition, there are other factors, such as facial features and hair texture which may betray a suspicion of Negroid origin. On the other hand, we are all familiar with persons one or both of whose parents are unquestionably Coloured but who are indistinguishable from typically White persons. Conversely, a dark-skinned, flat-nosed, coarse-haired, thick-lipped Caucasian could on occasions be mistaken for a Coloured person.

This leads us to the question: Which of us can claim to be of 'pure' race? The distribution of the blood groups among the various peoples of the world certainly indicates that no 'pure' races exist on earth to-day. The Caucasians themselves appear to be half proto-European, half Mongoloid, with a dash of Negroid strains.

Where, for purposes of legal classification, the question arises whether a person is White, Coloured, Negroid or Asiatic, the policeman and the tram conductor, unencumbered by biological lore, can make an assessment with greater conviction, and certainly with fewer reservations, than can the geneticist or anthropologist. Indeed, the law being traditionally intolerant of uncertainty in matters of definition, the evidence of the scientist on the subject of race can only prove an embarrassment to the Courts if not to himself.

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The Identification of Skeletal Remains

In Regina *versus* K and Another

The Howick Falls Murder Case

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Identification of a body which has been mutilated or burned beyond recognition may present considerable difficulty.

One of the techniques for establishing identity in such cases consists in comparing the dimensions of the skull of the deceased person with those of the head of the missing person as measured on a life-size enlargement of a photograph. A life-size enlargement is only possible if some object present on the photograph can be enlarged to its original size with certainty and be used as a scale.

Glaister and Brash[1] introduced this technique and established the identity of 2 missing women. In each case they superimposed a photograph of the skull of the deceased person on a life-size enlargement of a photograph of the missing person. One of the murdered women in this case had a photograph taken some years before she was murdered. She wore a diamond tiara at the time. Both the photograph and the tiara were recovered. One of the diamonds of the tiara was used as a scale to obtain a life-size enlargement of the photograph. The other woman was photographed near a gate. The photograph was recovered and the gate was used as a scale.

Gordon and Drennan[2] adopted the same technique in the identification of the remains of a person found in a burnt-out car. The life-size enlargement of a photograph of the missing person was obtained by making use of the pattern on a tie which was worn at the time when the photograph was taken, and which was recovered.

Simpson[3] investigated a case of a similar nature. He was unable to make a life-size enlargement of the photograph in his possession since there was no object which he could use as a scale for exact measurement. He obtained his enlargement by making the width between the orbits correspond with

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that of the skull. He was able to conclude that no dissimilarity was present between the skull and the photograph.

In the present case, identity was established by making use of the skull proportions and, in addition, certain facial characteristics peculiar to the missing person were used. No knowledge of the facial dimensions was obtainable since the photograph of the missing person was too indistinct.

History

On 16 February 1952 the decomposed body of an adult Indian male was recovered from the pool at the bottom of Howick Falls, Natal. The district surgeon who inspected the body was unable to determine the cause of death. The identity was not established before burial.

On 12 March 1952 the police received information that the body was that of an Indian known as 'Passing Show'. It was stated that on the evening of 12 February 1952 the deceased left Durban by car in the company of 4 other Indians and a European. On the way to Howick he was assaulted by his companions, put into the boot of the car and later that night thrown into the flooded river immediately above Howick Falls.

On the basis of this information the 4 Indians and the European were arrested. Three of the Indians turned Queen's evidence and the other 2 men were charged with murder.

After the arrest of the accused, the body found at Howick Falls was exhumed to try to establish the cause of death.

Subsequently uncertainty arose about the identity of the exhumed body, as it was suggested that the body found in the pool of the Falls was not that of 'Passing Show'. The body was therefore re-exhumed. A series of investigations were then undertaken to establish the identity of the deceased. On the completion of these investigations the police were informed that there was a strong probability that the body of the deceased was that of 'Passing Show'.

At the trial the Crown alleged that the 6 men, including the deceased, were members of a house-breaking and dagga-dealing gang. Following a disagreement about the distribution of money in their possession, the deceased threatened to expose the activities of the gang. The 2 accused then planned to murder him. On the assumption that the money would be given to him, the deceased was persuaded to go with the 5 men to Pietermaritzburg on the evening of the murder. After they had travelled some distance the car was stopped and the Indian accused instructed the deceased to get out of the car. The deceased resisted and in the ensuing struggle he was struck by the European with the butt of an automatic pistol. The deceased lost consciousness and the accused believed that they had killed him. He was put in the boot of the car and the journey was continued. On the way the deceased recovered consciousness and he was again assaulted. It was alleged that he was hit on the left side of the head

and face and that he sustained injuries in the region of the heart and the lumbar region. He was again put into the boot of the car, taken to Howick Falls and thrown into the river immediately above the Falls.

The accused were tried before a judge and 2 assessors, were found guilty of murder and sentenced to death.

Post-Mortem Findings

The first exhumation was carried out on 24 March 1952. Several wounds of the soft tissues of the head and face were found. Owing to putrefactive changes, no opinion could be given about the condition of the rest of the soft tissues. A comminuted fracture, involving the left temporo-parietal region and the left maxillary and zygomatic bones, was observed. The left humerus, several ribs, the spinous process of the seventh cervical vertebra and some of the spinous and transverse processes of the lumbar vertebrae were fractured.

The second exhumation was undertaken on 9 May 1952. A light moustache, covering the whole of the length of the upper lip, was still recognizable. The moustache and the hair found at the bottom of the coffin, in the region of the head, were black in colour. No further identifying features could be found.

The skull and the mandible, the upper lip, an area of skin from the right side of the skull and right cheek, bones of the hands and feet as well as the long bones of both arms and the left lower limb were retained for further investigation.

Identification

In this case the race, height and sex were known and the only factors to be determined were the age and the personal identity.

Determination of Age. The epiphyses of the long bones were fused to the shafts and ossification was present in the cartilage plate between the basilar part of the occipital bone and the body of the sphenoid bone. These findings suggested that the deceased was over the age of 23 years.[4] The sutures of the vault of the skull were not obliterated. Since this obliteration commences between the ages of 20 and 30, the upper age limit was taken to be 30 years. Relatives stated that the deceased was approximately 29 years old.

Personal Identity. A full-length portrait of the missing person, enlarged from a small snap, was recovered (Fig. 1). The snap could not be found. None of the clothing worn at the time when the snap was taken could be traced nor was there any other object present on the portrait which could be used as a scale. It was therefore impossible to enlarge the portrait to life size.

Although the portrait was indistinct and touched up, certain facial peculiarities could be noted and the facial proportions could be determined.

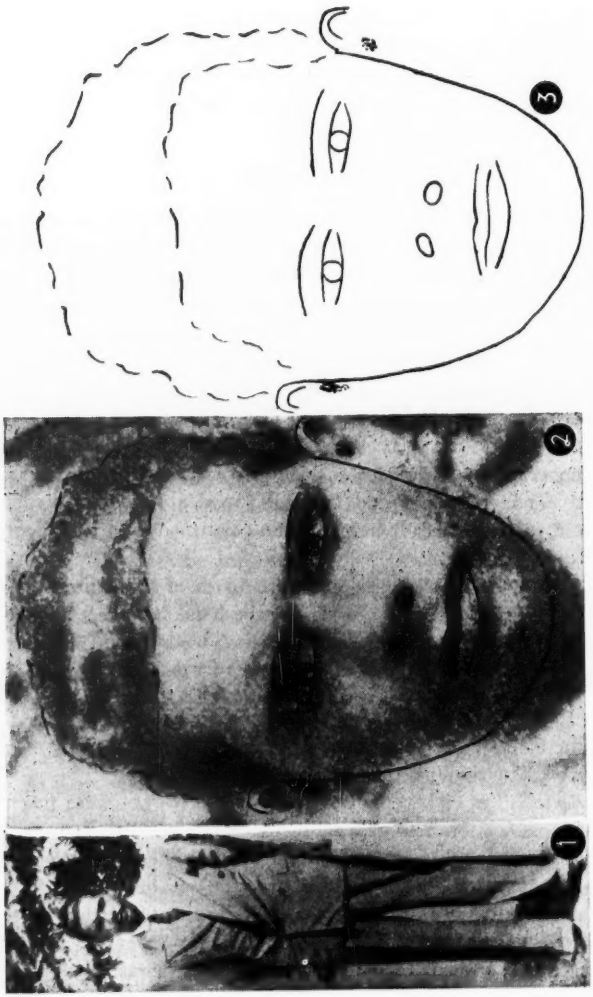

Fig. 1. Full length portrait of the missing person recovered by the police.
Fig. 2. Enlargement of the head of the portrait with certain structures outlined.
Fig. 3. Tracing of the features outlined on Fig. 2.

It was therefore decided to try to establish identity by comparing these facial proportions and peculiarities with those of the skull of the deceased.

The head of the portrait was enlarged to a size convenient for work. Certain structures, important in studying the facial proportions, were outlined on the enlargement (Fig. 2). These outlines were traced on to paper (Fig. 3).

It was noted on the enlargement that the distance between the left eye and left ear was shorter than the corresponding distance on the right side,

indicating that the head was turned slightly to the left.

The position of the head, in the horizontal and vertical planes, was estimated and the skull and the mandible of the deceased mounted, as accurately as possible, in the same position and photographed. The print of the skull was made to a size slightly smaller than that of the enlargement of the head allowing for the thickness of the covering soft tissues (Fig. 4).

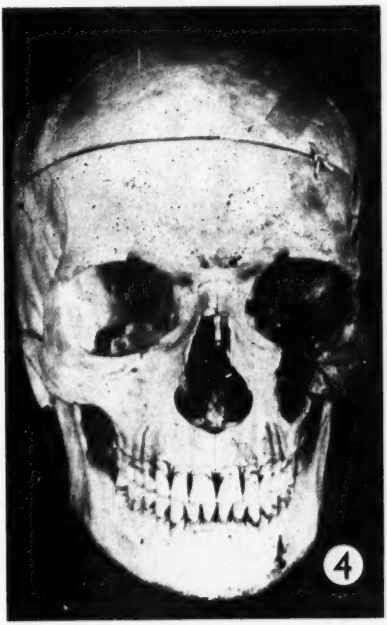

Fig. 4. Photograph of the skull.

Certain features of the skull, comparable with the features indicated on the portrait of the missing person, were outlined and traced on to paper (Fig. 5).

The 2 tracings were superimposed (Fig. 6) and compared. It was noted that the contour of the head of the missing person was of the same shape as that of the skull, the eyes fitted into the orbital cavities of the skull and the mouth overlay the teeth. These findings indicated that the portrait and the skull could be that of the same person.

On examining the portrait of the missing man, the impression was gained that the nose was slightly deviated to his right side. An attempt was made to prove that this observation was correct. A line drawn from the nasion to the point midway between the 2 external nasal apertures and extended downward was seen to lie to the right side of the philtrum of the upper

lip (Fig. 7). In a person with a nose which is not deviated, a line such as this should fall to the left of the philtrum when the head is turned to the left. This observation indicated that the nose of the missing person was deviated to the right side.

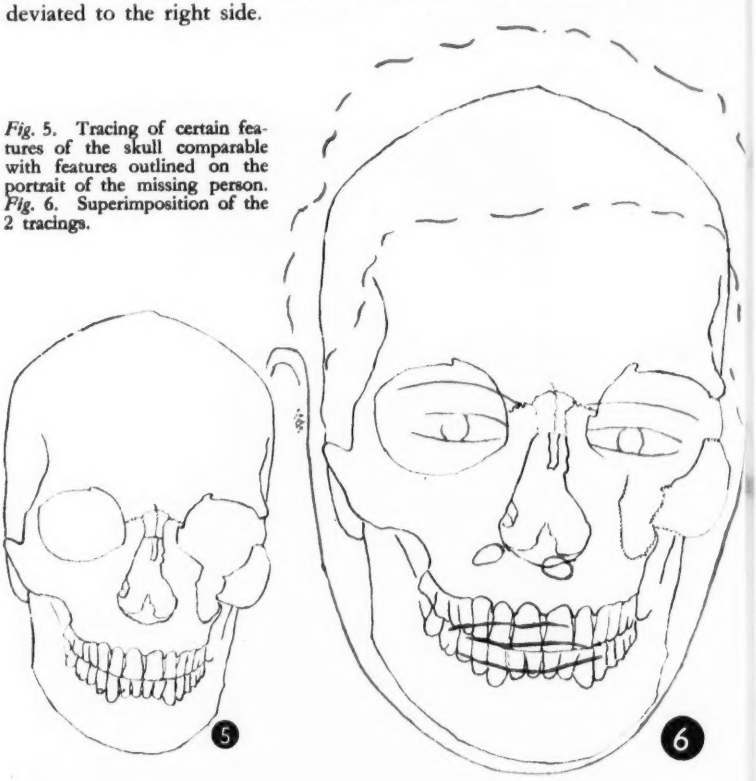

Fig. 5. Tracing of certain features of the skull comparable with features outlined on the portrait of the missing person.
Fig. 6. Superimposition of the 2 tracings.

Examination of the skull revealed an incompletely ossified vertical fissured fracture of the wing of the left maxillary bone with deviation of the fractured part of the bone to the right side. In addition, there was an old fracture of the right nasal bone and the nasal septum was deviated to the left. These findings indicated that the nose of the deceased was deviated to the right. The latter observation was regarded as a strong point in favour of the assertion that the body recovered at Howick Falls was that of the missing person.

The result of all the investigations indicated that the deceased was an adult male of the same age group as that of the missing person, with a head of the same proportions and in addition with a nose deviated to the same side as that of the missing person. It therefore seemed reasonable

to conclude that there was a strong probability that the body of the deceased was that of the missing person.

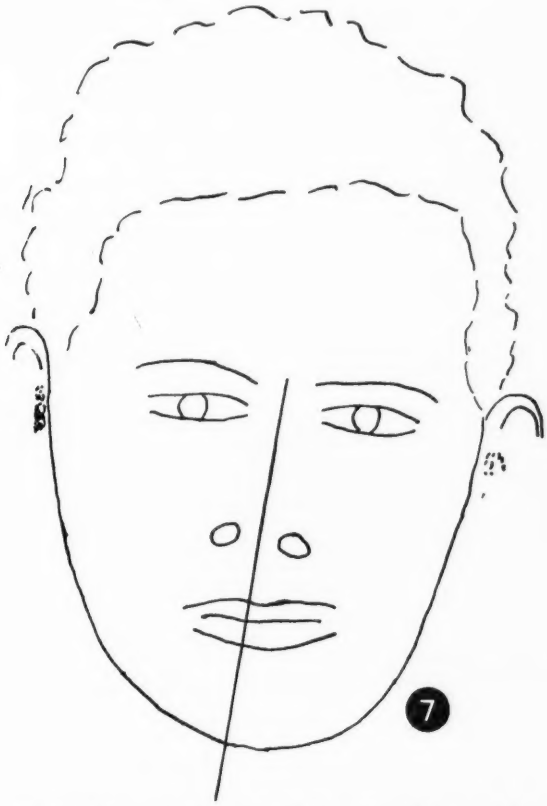

Fig. 7. Tracing showing that the line connecting the nasion and the point midway between the 2 external nasal apertures lies to the right of the philtrum of the upper lip.

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MEDICO-LEGAL CASE BOOK

I: Dentures and Individual Identification

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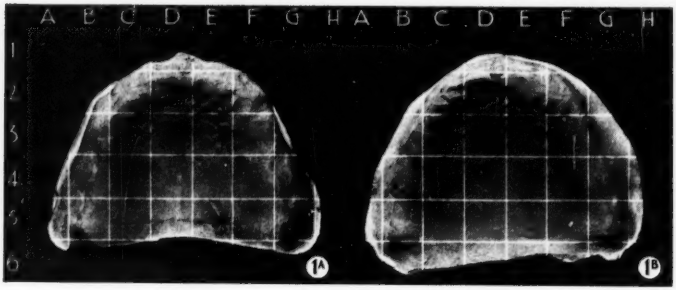
Figs. 1A and 1B formed an exhibit in the case of Regina *vs.* Karusseit (Pietersburg Circuit, 7—11 April 1953). The accused was charged with murder.

The evidence against the accused was completely circumstantial. Fig. 1A



is a photograph of a plaster cast of an upper denture, believed to have been that of the deceased. It was found 5 months after the murder concealed in a lavatory at the Pietersburg Police Station.

Fig. 1B is a photograph of a plaster cast of a wax impression of the deceased's palate, made 3 days after death.

The corresponding depressions and elevations can be compared. Note particularly squares 2C—2F and 3C—3F, all of which correspond completely in the illustrations. The photographs prove that the denture must have been that of the deceased.

The fact that the accused had not been searched until he had been at the Police Station on the day after the murder for about 2 hours, that he would have had an opportunity of concealing the denture in the lavatory and that, when found, it was covered with dust, were links in the chain of events connecting him with the body.

The accused was sentenced to death.

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Sudden or Unexpected Deaths in Infancy

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Many children die suddenly or unexpectedly in their beds from natural disease. In Melbourne alone, during 1952, there were 64 such deaths. This gives rise to one of the most difficult problems the forensic pathologist has to face, viz. to determine satisfactorily the cause of death in a case of this nature, especially if the child is found dead face downwards or beneath the bed clothes or in bed with his parents.

In his search for the cause of death the pathologist must be prepared to carry out a thorough and painstaking post-mortem examination, including not only the usual naked-eye inspection of organs, but routine microscopy and bacteriological and serological investigations as well. After the most searching autopsy, where there is no doubt that death was due to natural disease, the pathologist, occasionally, may not be able to find the cause.

In the majority of these children a careful search will show a satisfactory cause for death and in some instances the autopsy will offer little difficulty, as Case 1 illustrates.

Case 1: A male child, aged 2½ years, said to be in good health and apparently quite well after his breakfast, was taken by his mother to Church that Sunday morning. He appeared to be his normal self until after lunch that day when he complained of feeling sick and vomited during the afternoon. Later in the afternoon he became rapidly worse and before medical attention could be procured the boy died. Autopsy showed that he was suffering from acute meningitis. This condition was suspected on naked-eye inspection and confirmed by microscopy. This boy also had a small, irregular calculus impacted in the cystic duct, causing obstruction and distension of the gall bladder with white bile. There were also numerous tiny yellowish calculi present in the gall bladder. There was no jaundice. There was no evidence of acholuric jaundice, nor any other abnormality.

It should be clearly appreciated, however, that nothing of note may be visible on ordinary naked-eye examination of the organs at autopsy, yet striking changes may be found in one or more organs on microscopic investigation (Case 2).

Case 2: R. A., a male baby aged 11 months, who was said to have been in good health, awakened at 3 o'clock one morning and appeared to be restless. About 6 a.m. he was given his usual milk feed which he drank and then appeared to go to sleep. When his father woke up at 7 a.m. he found the child dead, lying face downwards in the bedding.

Autopsy showed the body of a well-developed, well-nourished child. The heart was a little dilated and the muscle rather pale. There was nothing else of note to be seen apart from slightly enlarged glands at the hilus of each lung.

Microscopic examination showed some infiltration of the peribronchial tissues with lymphocytes and mononuclear cells. There was acute non-specific myocarditis with gross infiltration of the myocardium with round cells, endothelial cells, eosinophils, macrophages and some giant cells as well as patchy destruction of muscle fibres. These lesions were not suspected before microscopy.

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If this autopsy had not been done and, if done, had not proceeded beyond the macroscopic stage, the diagnosis would probably have been 'suffocation'.

In some cases nothing definite will be found on naked-eye examination and only slight or minimal changes on microscopic examination, so that the pathologist is left wondering whether what he has found is sufficient to cause death (Case 3).

Case 3: A mother, a trained nurse, put her baby (a male child aged 8 months) to sleep on his back in his pram on the front verandah of her home. Half an hour later she went to see the baby and found him dead in his pram on his back, face uncovered, bed clothes undisturbed, exactly as she had left him. Autopsy showed only a mild bronchitis and nothing else that could be regarded as significant.

It seemed quite obvious that this baby died of disease but the microscopic findings were not sufficiently convincing to account for death. It is in instances like this that the importance attached by different observers to the microscopic changes detected will vary, especially when it is remembered that in a control series minor bronchitic changes are sometimes to be observed.

Nothing definite, macroscopically or microscopically, may be found after a careful search at autopsy to explain a child's death (Case 4).

Case 4: A female child aged 3 years and 4 months was thought to be a little flushed about the face one afternoon. She had her usual evening meal but shortly afterwards vomited. She went to bed at 7.15 p.m. and was given three-quarters of an 'Aspro'. The following morning her father heard her breathing heavily and went for medical aid. He returned with a doctor 5 minutes later but the girl was dead. Post-mortem examination did not show anything to explain her death satisfactorily. No bacteriological examination was carried out in this girl.

Autopsy Findings in a Series of 320 Sudden Deaths in Infancy

In the past 6 years we have investigated 320 instances of sudden or unexpected deaths in childhood. These were all coroner's cases. The series includes all infants and children aged less than 14 years whose bodies (in a few cases organs only) were brought to the Melbourne City Morgue for investigation. The majority of the children were found dead in bed; they were not thought to be seriously ill; they had received no medical attention and death was sudden or unexpected. Some of the children in the series were known to be ill but died before they reached hospital or before the nature of the illness could be established by the doctor in attendance. In each instance an autopsy was ordered by the coroner. Microscopic investigation of the organs was carried out as a routine and in 22 of these children a special bacteriological study was made[1]. The pathological conditions found are shown in Table I.

Discussion

Sex Distribution: There were more deaths in male than in female children, the ratio being approximately 11 : 7.

TABLE I: PATHOLOGICAL LESIONS DETECTED

Disease	*Number of Times Found*	*Disease*	*Number of Times Found*
Acute infection in the respiratory tract	133	Diabetes	3
Meningitis	28	Epilepsy	3
Meningococcal septicaemia	25	Acute pericarditis	2
Acute myocarditis	27	Hepatitis	3
Otitis media	18	Acute empyema	2
Undetermined	18	Hydronephrosis	2
Congenital heart disease	13	Polycystic kidney	1
Atelectasis (marked)	12	Nephritis	3
Acute pulmonary oedema	10	Encephalitis	4
Septicaemia (shown by culture)	9	Resorption atelectasis (hyaline membrane)	2
Enlarged heart	9	Diffuse haemorrhagic pneumonia	2
Bulbar poliomyelitis	8	Pulmonary tuberculosis	2
Malnutrition	7	Intussusception	2
Acute entero-colitis	7	Cleft palate, hare lip	2
Intestinal obstruction	6	Myocardial fibrosis	2
Subarachnoid haemorrhage (non-traumatic)	4	Foetal endocarditis	2
Intracranial haemorrhage (traumatic)	3	Haemorrhagic suprarenals (non-meningococcal)	2
Lobar pneumonia	4	Cerebral tumour	2
Erythroblastosis foetalis	4	Mongolism	2
Fibrocystic pancreas	4	Hydrocephalus	2
Acute pyelonephritis	3	Lipoid pneumonia	1
Fatty liver	3	Purpura	2
Acute pleurisy	3	Acute appendicitis	1
Hydro-ureter	3	Fractured skull	1
Congenital syphilis	1	General peritonitis	1
Diaphragmatic hernia	1	Acute pancreatitis	1
Acute tonsillitis	1	Congenital cystic lung	1
Glycogen storage disease	1	Polyarteritis nodosa	1
Hyperplasia of the islets of Langerhans	1	Status asthmaticus	1
Bronchiectasis	3	Cholelithiasis	1
		Cerebral atherosclerosis	1
		Advanced thrush	1

Age Incidence: The number of deaths in each age group is set out in Table II.

TABLE II: AGE GROUPS

Months Under 6	*Months 6–12*	*Years 1–2*	*Years 2–3*	*Years 3–4*	*Years 4–5*	*Years 5–6*	*Years 6–7*	*Years 7–8*	*Years 8–9*	*Years 9–10*	*Years 10–11*	*Years 11–12*	*Years 12–13*	*Years 13–14*
164	52	25	26	15	4	6	7	5	1	3	3	1	0	5

Approximately half of the deaths occurred in infants less than 6 months of age. This series includes very few babies who died in the first 2 weeks of life, as death usually occurs during that period in hospital. Very few deaths occurring in this age period are reported to the coroner in this city, the investigation of stillbirths and neonatal deaths being carried out usually at maternity institutions. Two-thirds of the deaths occurred in infants less than one year old and $\frac{2}{3}$ of the deaths occurred in children less than 3 years of age. After the third year of life sudden death in childhood

has not been common from natural causes. Sudden deaths in the older children are more likely to take place if there is some particular infection prevalent in the community at the time, e.g. poliomyelitis or meningococcal septicaemia. Of the 8 cases of bulbar poliomyelitis recorded in this series, 6 were in children more than 3 years old and they occurred when poliomyelitis was prevalent in the community. Acute pulmonary oedema was the outstanding finding in these sudden deaths where the brain stem was involved.

History. Although it was commonly stated by the parents at the time of death of the child that he was not ill and appeared to be quite well, a follow-up and enquiry into 140 of these deaths showed that signs and symptoms of illness were exhibited by 77 of the children. Where death occurs the parents are, as a rule, so affected that a reliable history is not obtained, especially where the statement is taken from the mother by a policeman. A police officer who visits the home where the child has been found dead is unlikely to ask whether anyone else has been ill in the house or e.g. whether the child has been in recent contact with someone else suffering from a cold or some other respiratory complaint. We have been impressed by the frequency with which the deceased children did have symptoms before death suggesting that death was due to natural disease. In some cases we have known that the deceased child had a temperature of 103° F or even 105° F before death and were not able to demonstrate anything very convincing at autopsy to explain the rapid death.

Position of the Child when found Dead. A careful check of 70 consecutive cases in Melbourne showed that 10 of the children were found dead face downwards on the bedding (including pillows) or under the bed clothes or in bed with the parents. One of the babies was lying dead in his pram on his back with the family cat curled up asleep on his face. In these 10 cases the question of accidental suffocation or overlaying had to be considered. Of these 70 cases it seemed quite clear that at least 86% of the deaths were caused naturally. Many young children adopt the prone position to sleep; they prefer to lie face downwards with the body flexed and the knees well drawn up. If this is the position of choice of many children and if some of them should die of unsuspected disease, it may be anticipated that some infants will be found dead face downwards and in a pillow, if there is one. A mother may take her sick child into bed to sleep with her, she may take a distressed crying child into bed with her during the night, and if that child should die in bed with her during the night, it does not follow, without adequate post-mortem investigation, that death was due to overlaying.

Acute Infection in the Respiratory Tract. Evidence of acute infection in the respiratory tract was found in 133 of these children. This is the commonest and most outstanding pathological lesion encountered in sudden death in infancy. The term here has been used to cover such conditions

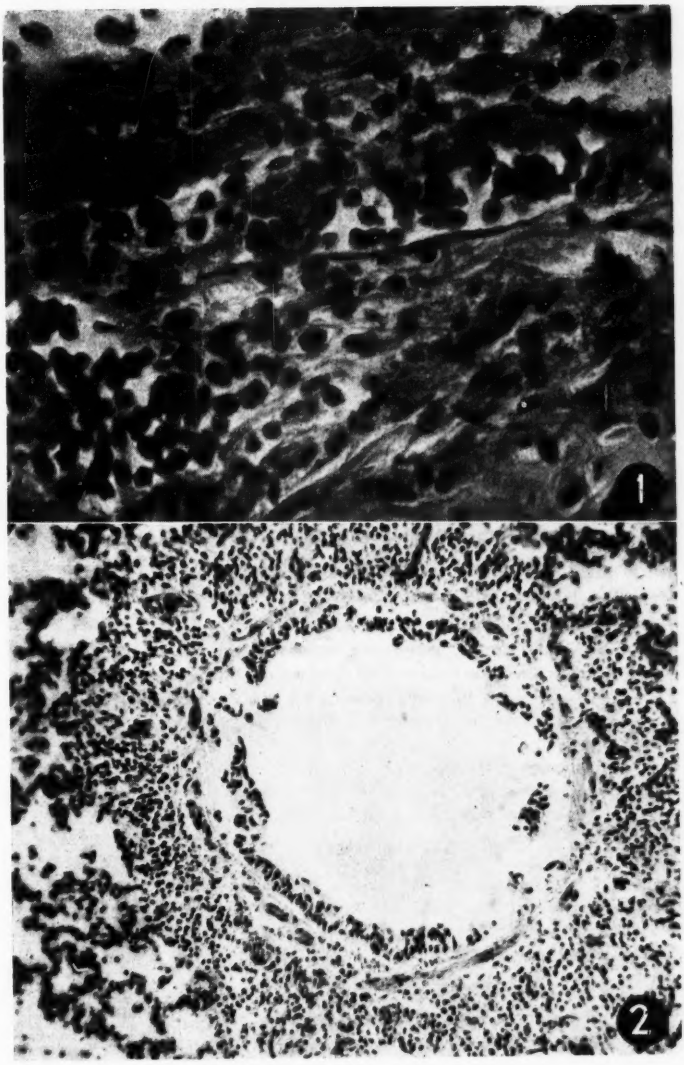

Fig. 1. Photomicrograph of the bronchus of a 13-month-old baby found dead face downwards on the bedding. It was assumed that this child had suffocated. The bronchial wall is heavily infiltrated with polymorphonuclear leucocytes. There was also fibro-cystic disease of the pancreas.

As 5 children out of 10 in this family died in infancy, it was assumed there was parental neglect and it was proposed to proceed against the parents. The other children probably also suffered from fibro-cystic disease.

Fig. 2. Bronchiolitis. This baby was found dead beneath the bed clothes and suffocation was again assumed.

as acute inflammation of the epiglottis, acute laryngitis, acute tracheitis, acute bronchitis, acute laryngo-tracheo-bronchitis, bronchiolitis and bronchopneumonia. Lobar pneumonia was rarely encountered and is listed separately 4 times. It is interesting to note that in one baby with frank lobar pneumonia the mother stated that her baby was quite well; she had suspected no illness before death.

Acute infection in the respiratory tract may be overlooked easily on naked-eye examination at autopsy and particularly so on microscopic investigation if the lung parenchyma is the only portion selected for study. Tissue from the root of the lung showing the larger bronchi is very useful for routine sections, due allowance being made for the normal presence of lymphoid tissue in the interpretation of the findings. Oedema of the alveolar sacs, vascular engorgement, thickening of the interalveolar walls, some slight accumulation of round cells and a few polymorphonuclear cells in the walls, are common findings, but if the bronchial tree is not examined in these cases, clear-cut evidence of acute infection may be overlooked (Figs. 1 and 2). Acute epiglottitis, acute laryngitis and laryngo-tracheo-bronchitis may bring about extremely rapid death from intense inflammatory swelling and obstruction to the airway and these conditions may be accompanied by an overwhelming septicaemia.

Much less marked inflammatory change in the upper part of the bronchial tree may soon be followed by death (Case 5).

Case 5: A male child aged 10 months, the son of a doctor, was examined by a paediatrician at 4 p.m. one afternoon for a routine check-up of his feeding. He was not thought to be ill. At 7 o'clock the following morning his mother found him face downwards dead in his cot. Although this baby could walk his mother concluded that he must have suffocated accidentally.

There was reddening of the mucous membrane of the larynx, trachea and bronchi and microscopy showed some denudation of the respiratory epithelium, engorgement of the blood vessels, oedema and infiltration of the bronchial mucosa by mononuclear cells Further enquiries showed that the baby had had a running nose for 24 hours and that 2 older children in the house were recovering from acute respiratory tract infections. One of the sisters had been very ill. A bacteriological study was carried out on the dead baby including a search for influenza virus, but no significant organism was isolated.

Acute Myocarditis. Non-specific acute myocarditis, a much neglected disease, is a significant cause of sudden death in infancy. This condition needs to be emphasized as a clinical and pathological entity and especially as a cause of sudden death. Acute myocarditis was found 27 times in this series, while only one block of heart muscle (sometimes 2) was examined as a routine. The term acute myocarditis is used here in the sense of a non-specific acute inflammatory reaction involving any part of the myocardium, muscle or interstitial tissue. There may be very little to see at autopsy and very little on naked-eye examination to indicate its presence and unless the heart is always examined microscopically, many cases will be missed. Every part of the myocardium may not be involved and unless more than one block of heart muscle is available for study

the condition may not be discovered. Inflammatory changes may be pronounced in one part of the heart wall and absent from the adjacent muscle, e.g. in one person 2 adjacent blocks of tissue cut from the left ventricle in a fatal case of poliomyelitis were examined. In one there were insignificant changes; in the other there was frank myocarditis.

Inflammation of the myocardium occurs as a complication or as part of the pathological picture in a wide variety of diseases, or it may be found as an isolated entity. It may occur *in utero*. In one heart which we had the opportunity to study in the case of a baby dying on the first day of life, there were wide-spread changes in the myocardium. Two episodes of acute non-specific myocarditis have been recognized in the past 6 years among the coroner's cases at the Melbourne City Morgue. The first group of cases occurred in 1949—1950 at the same time as an outbreak of poliomyelitis in the community. After an interval the condition was found again in 1952 in another group of cases, suggesting that the disease occurs as a periodic infection. It has also been noted that acute non-specific myocarditis occurs in association with inflammatory changes in the meninges (meningo-myocarditis) and in association with inflammatory changes in the upper part of the respiratory tract.

Case 6: R. B., aged 12 days, was examined by a doctor at 11 o'clock one morning because he was off his food. He was revisited at 6 p.m. that day just after he had vomited and apparently asphyxiated. It was assumed that aspiration of vomitus had caused death from asphyxia.

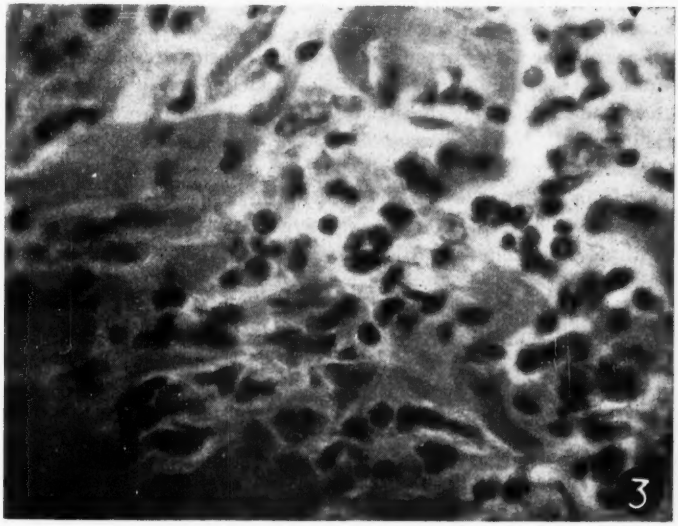

Fig. 3. Acute non-specific myocarditis. Infiltration of the myocardium with inflammatory cells.

Autopsy showed a child who was underweight, the heart was a little enlarged, the muscle pale and the foramen ovale and ductus arteriosus were still patent. The trachea and bronchi contained frothy fluid, no food. There were petechial haemorrhages on the surfaces of the lungs.

Microscopic examination showed oedema, capillary congestion, haemorrhages, cloudy swelling and necrosis of heart muscle fibres. There was loss of striation, vacuolation and complete disappearance of other fibres. A lace-work appearance was left in some areas from disappearance of muscle substance. Heavy infiltration of the myocardium had occurred with polymorphonuclear leucocytes, eosinophils, macrophages and young fibroblasts (Fig. 3). No other microscopic changes in any other organs were detected.

Septicaemia. This is another significant cause of death in childhood, but one difficult of diagnosis, apart from meningococcal septicaemia, without bacteriological aid. It may be easily overlooked at autopsy (Case 7).

Case 7: S.A., a male child aged 4½ months, had been apparently well and was found dead in his pram with his face downwards in a pillow. Autopsy was undertaken about 10 hours after the body was discovered. On macroscopic examination the only abnormality was left otitis media and the presence of petechiae on the heart and lungs. On microscopic examination no definite abnormality was found. The pus from the left middle ear produced a heavy growth of *Streptococcus pneumoniae type IV* on culture. The heart blood also provided a scanty growth of *Streptococcus pneumoniae type IV*. The remaining cultures were not significant. The finding of the same serological type of pneumococcus in the middle ear and the heart blood strongly suggests that the child suffered an invasion of his blood stream from the middle ear probably some time before death.

In 22 consecutive cases in this series bacteriological examination was undertaken *post mortem*. Culture of the heart blood gave positive results in 6 instances; it was negative in the remaining 16 cases. Where the heart blood culture was positive, *Staphylococcus pyogenes*, *Escherichia coli*, *Haemophilus influenzae type B* and *Streptococcus pneumoniae type IV*, were the organisms obtained. In 4 of the cases in which a positive result was obtained on heart blood culture, clear evidence of bacterial spread from a defined focus was obtained among these 22 cases. Only 2 were of proven meningococcal infection. *Neisseria meningitidis type I* was cultured from a pericardial exudate and numerous intracellular meningococci were demonstrated in a smear from a skin rash. Seven others in this specially studied group of 22 cases showed features strongly suggestive of a meningococcal septicaemia but with negative cultures. It is of interest that from 2 of these influenza virus was isolated.

Apart from this bacteriological study of 22 consecutive cases, in the whole series a diagnosis of meningococcal septicaemia was made 25 times. This diagnosis was based on a typical petechial skin rash, oedema about the suprarenals and macroscopically haemorrhagic adrenal glands; meningococcal septicaemia was present in the community at the time. In our experience meningococcal septicaemia is a very important cause of sudden or unexpected death in infancy.

Meningitis. There were 28 cases of meningitis in this series, but no study was made of these cases bacteriologically. The condition may be recognized easily at autopsy, but in some cases the diagnosis depends on microscopic

examination. The shortness of the duration of the illness and the importance of microscopic investigation are emphasized by the death of the child mentioned in Case 1.

As part of the search for the cause of death the bodies of all the infants were weighed. A prominent feature was the frequency with which the body was markedly underweight.

An interesting feature in our cases was the infrequency of gastro-enteritis or entero-colitis as a cause of death. It has not been an important cause of sudden death in infancy in Melbourne.

Controls. In interpreting particularly the microscopic appearance of organs in infancy and childhood, the forensic pathologist has the necessary opportunity for establishing his own criteria of normality. Death from unnatural causes such as drowning, motor car accidents, etc. provides the necessary material for comparison. Whenever suitable material has been available in the Morgue it has been used for a control series. Mild degrees of infection in the bronchial tree are encountered in the control cases in childhood. Apart from the bronchial tree there is little or no difficulty with other organs. It does not follow because there is a mild degree of bronchitis that it is significant in the death of the child. It may mislead the investigator and lead him away from the real cause of death.

It is no reflection on the pathologist (especially if he is called upon to carry out his work in the usually poorly equipped Government Morgue) if he is unable to find a satisfactory cause of death. He should not hesitate to tell the coroner so.

Conclusions

A study of sudden deaths in infancy and young children clearly indicates that no matter what the position of the child in bed at the time of death, death is probably due to natural disease.

A thorough post-mortem examination is usually necessary to establish a satisfactory cause of death. In carrying out these autopsies the evidence of disease may be overlooked easily. Routine microscopy and bacteriological examination may reveal disease not suspected with the naked eye.

Minor degrees of bronchitis may be found in control cases and death is not necessarily due to this. Petechial haemorrhages on the heart, lungs and thymus and a picture of asphyxial death may be a conspicuous feature when death has clearly resulted from natural disease.

No coroner should accept a diagnosis of accidental suffocation or overlaying in the absence of a thorough post-mortem investigation.

Reference

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The Use of X-Rays as an Aid to Medico-Legal Investigation*

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In isolated communities, the Medical Examiner frequently sees cases in which a complete medico-legal autopsy may be highly desirable, but at the same time impracticable for various reasons.

My territory is Southampton County, located in Tidewater, Virginia, on the edge of the Dismal Swamp, in the Smithfield Ham and the Planter's Peanut belt. Total area: 606 square miles. Population: approximately 30,000 (60% Coloured; 40% White). Our population is almost static with 97% native born. It is a rural community with only one town of 5,000, the next largest town having 800. We are primarily agricultural—peanuts, Smithfield hogs, cotton, corn and tobacco. Our second largest industry is lumber and wood-pulp products.

In the Commonwealth of Virginia under the present Medical Examiner system, medico-legal autopsies are accomplished by 25 designated qualified pathologists in 20 different localities. Those nearest to my county are in Norfolk, a distance of 50 miles, and in Richmond, a distance of 75 miles. Because of the time necessary for transportation, it becomes most important that we carefully select or screen our cases which will receive complete medico-legal autopsy and we believe that this screening should be done locally.

Because of this situation and my primary interest in diagnostic X-ray work, we have found that with increasing experience in medico-legal investigation, a rather surprising percentage of our cases can be screened with the aid of X-rays and laboratory facilities which are available at our local, community-type, 75-bed hospital. Available X-ray equipment consists of one stationary 100 Ma General Electric R-36 unit which has been in continuous operation for 16 years and a 30 Ma bedside portable unit.

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For several years, my group in the Raiford Memorial Hospital has been particularly interested in gaining more knowledge about the problems related to the identification of single or large numbers of badly burned human bodies. My county is well within the perimeter of a theoretical hydrogen-bomb blast which might be centred on the Norfolk, Newport News, Hampton Roads area. We believe that the monumental and pioneer reports of the Toronto, Canada, Medical Identification Committee for the Noronic Disaster should receive careful study and attention. My first 2 cases illustrate isolated examples of the application of X-ray studies on human bodies which have been burned beyond any hope of visual identification.

Fig. 1. (Case 1). Bony remains of male infant (6 months) after separation from debris.

Case 1: (*Eaton Baby*). A pillow case was found filled with suspected human remains and surrounding charcoal scooped up with a shovel from

the smouldering remains of a 3-room wooden shanty. The family of this decedent reported that this was a 6-month-old, Coloured male who had been overlooked by the rest of the family in a mad scramble for safety when their home was deliberately set on fire by the father of the household in a drink-maddened attempt to wipe out his entire family. Dogs, cats and pigs were known to have been in or under the house at the time of the conflagration. Differentiation between human and animal remains was therefore the primary problem.

Wood charcoal is completely transparent to X-rays, which revealed definite evidence of human remains, so a careful selection of fragments was carried out and the charred human remains were placed in a position to reconstruct the body of this infant.

Fig. 1 shows the result of the separation. The lower cervical and upper thoracic area was thought to be a portion of the pelvic structures. Some of the bony structures were completely carbonized and could be very easily crushed into dust by rubbing between the fingers. I would like to call

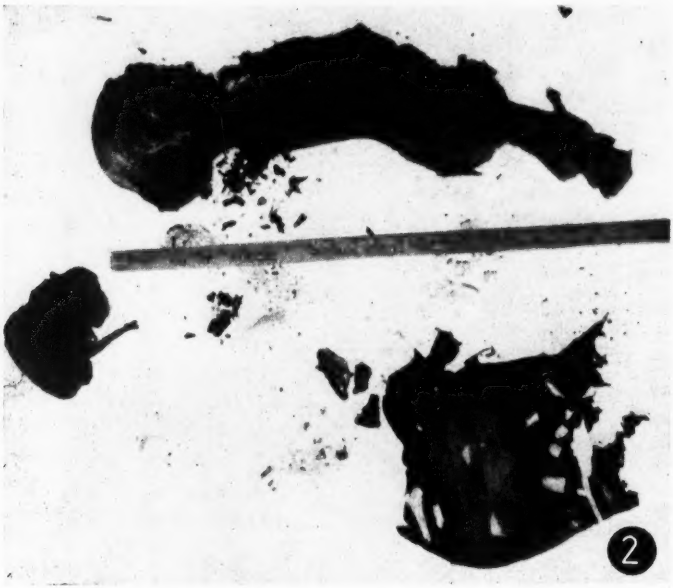

Fig. 2. (Case 2). Photograph of charred remains of a 21-month-old male. Visual identification impossible.

your attention to the remarkable state of preservation of the bony trabeculation. The chambers of the heart are still intact. It is still possible in such a case to obtain enough blood from the chambers of the heart to determine

death from carbon monoxide, or in the absence of carbon monoxide to determine that this decedent was dead at the time of conflagration.

Case 2: R. C. P. (Peewee Person). Fig. 2 is a photograph of the charred remains of a Coloured male, age 21 months. This baby lived in New York but was visiting its great-grandmother in my county when, for some unexplained reason, the 2-story frame dwelling caught on fire and this decedent was trapped. We were suspicious of arson, but it was never

Fig. 3. (Case 2). Lateral view of the skull.

proven. I think that you can clearly see that visual identification of this body would be impossible. At the time of death, there was reason to doubt all of the story which was given me by the alleged relatives.

A lateral X-ray of the body showed that much of the bony structures of the spine (cervical, thoracic and lumbar) was visible. An A-P X-ray of the body showed the shrunken lungs and the very excellent bone detail of most of the remaining skeleton, as well as the unerupted teeth in the

small remaining fragment of the right side of the mandible. We believe that this degree of destruction of the human body is very characteristic of the usual findings.

A lateral view of the head (Fig. 3) showed the characteristic pattern of the burned skull after explosion of the cranium and the characteristic retraction of the scalp. Note again the unerupted teeth which could lead to fairly easy estimation of the age of decedent.

We believe that X-ray studies in this type of case are more important than is generally realized.

Probably of greatest practical help in the medico-legal investigation is the use of fluoroscopic and X-ray examination in the finding, location, and as an aid to removal of foreign bodies such as bullets, shotgun pellets, knife blades, ice picks and various other types of opaque lethal foreign bodies.

Fig. 4. (Case 3). X-ray films of the hand. Severe mutilation by hogs. Eight shot in soft tissues. This fluoroscopic find led to the solution of this case.

Case 3: B. F. C. A Coloured male, aged 57, was a retired storekeeper and landlord of considerable means. Known to be a severe cardiac, he was reported to have 'dropped dead of a heart attack while feeding the

hogs and the hogs dun et him up'. The body was not discovered until about 24 hours after death and a herd of approximately 75 hogs had severely mutilated the body. All exposed parts had been eaten and the body was covered with mire and offal. A very large hole had been eaten on the right side of the face. It was impossible to determine by gross examination that this was anything other than a natural death. Mainly because of curiosity and in an effort to record the amount of destruction of the body by the hogs, this decedent was placed on a fluoroscopic table for routine fluoroscopic screening.

Imagine our surprise when shot were found in the hand (Fig. 4). Note the extreme amount of soft tissue damage which we had supposed was due to mutilation by the hogs.

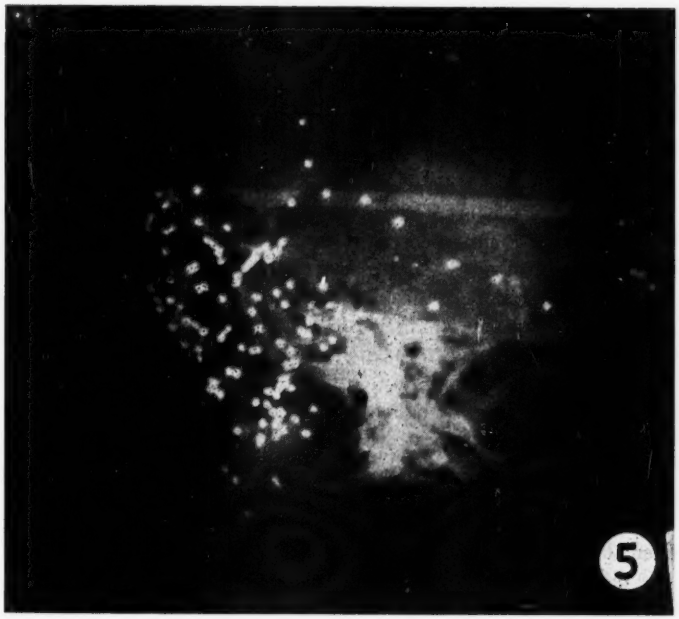

Fig. 5. (Case 3). X-ray of the skull showing shot.

The skull was still more impressive (Fig. 5). There were other films of the body which also showed shot, but following this discovery we were able to determine that this man had been shot at least 3 times from at least 3 different directions and probably from 3 different distances. It is our opinion that the third load of shot, fired from a very close range, apparently passed through the hand and into the skull. Of course, if this body had been found before mutilation by the hogs, shotgun wounds would have

been easily discernible. But for a high index of curiosity and the aid of the fluoroscope and X-rays the tenant of this old man's farm, who received 30 years for his murder, would never have been suspected. With fluoroscopic guidance, lead pellets were removed from the hand, skull and arm for presentation as Court evidence. These X-ray films along with others made during the investigation aided materially in presentation of this case in Criminal Court.

Case 4: P. G. F. In this case an A-P view of the skull showed the wound of entry of a bullet in the front of the forehead, just between the eyes and showed the location of the bullet in the posterior portion of the skull.

This was a White male, aged 67. He was a very wealthy landowner, a severe arthritic whose amorous attentions toward the wife of one of his tenants far exceeded his physical ability and who had been systematically fleeced of an amount in excess of an admitted $10,000 and who, upon learning of the unfaithfulness of the object of his attention sent a note to his lady-love stating his reasons for, and his intentions to, kill her and then himself. The woman testified: 'I finally talked him out of killing me and then he killed himself.' The alleged suicide was with a .22 Colt Woodsman target pistol and the decedent was found sitting on the bedroom floor of his lady-love, with the pistol in his right hand, a bullet wound in the centre of his forehead. Strangely enough the victim's hat and glasses were still in their accustomed positions and there were no powder burns on the forehead, hat or glasses. This man was known to be such a severe arthritic that he had been unable to tie his shoelaces, yet the Defence claimed that this man shot himself with a target pistol.

On a lateral film, an instrument inserted through the wound of entry was found to be far too short to reach the bullet, but it did show the direction of the path of the bullet. With fluoroscopic guidance, this bullet was easily removed through the hole of entry with a lithotrite and complete autopsy of the skull for recovery of the bullet was then unnecessary. In this case, the X-ray offered conclusive evidence in the controversy of suicide versus homicide and facilitated recovery of the lethal bullet. Incidentally, the threatening note written by decedent was placed in a casette along with driver's permit and other signed identification cards which were known to be in the decedent's handwriting and a photostatic copy of the known and questioned specimens of writing recorded on X-ray film with a flash exposure. Comparison and study determined that the threatening note was certainly written by the decedent.

Case 5: L. R. A Coloured male, aged 45, while on a week-end drinking party received very rough treatment from a group of his 'friends'. During the fracas, he was shot, beaten and cut. He was shot at 3 times, only one bullet hitting its mark, on the right side of the head. A probe was inserted in the path of the bullet. He was hit over the left side of the head with a heavy, blunt instrument and cut on the left side of the neck and jaw with the butt

end of a broken Coca-Cola bottle. Immediately after these injuries, he was transported across the State line by other 'friends' to a hospital in a neighbouring town in North Carolina where he died soon after admission and approximately 2 hours after his injuries. No Coroner's investigation was made of this death in North Carolina. The body was picked up by a Virginia undertaker, brought back into Virginia, and embalmed. Incidentally, transportation of unembalmed bodies across our State border is also illegal. Approximately 24 hours after death, the sheriff of my county called and asked for help in determining the cause of death and recovery of the bullets if they were the cause of death. Telephone inquiry to the Chief Medical Examiner's office authorized me to give the law enforcement agencies any help possible.

Fluoroscopic screening revealed only this one bullet with no clinical evidence to indicate other bullet injuries.

There was a very ragged laceration over the left side of the skull under which 2 definite fracture lines could be visualized on X-ray.

The fracture lines of the left side did not connect with the fracture lines incident to the passage of the bullet through the right side of the skull.

The third wound, a laceration at the level of the left angle of the mandible, as shown by probe, was proven to involve only skin and subcutaneous tissue to disprove the supposition that this also was a bullet wound.

The films, along with others incident to the case, when shown in Court, aided considerably in the conviction of one man for murder and the conviction of 2 others for attempted murder.

Case 6: H. T. A Coloured male, aged 26, was found at the scene of his death (Fig. 6) at 3 a.m. on a Sunday morning following a Saturday night birthday drinking party which was punctuated and enlivened by the firing of at least 3 and probably 5 different firearms. We were notified of the crime by the defendant in this case who claimed self-defence and who personally walked into the Police Headquarters of our town saying: 'I shot a man in the head. Here's the gun I shot him with and here's the gun he was shooting.'

Fluoroscopic examination readily revealed the site of entry and showed the location of the bullet. An A-P view showed more clearly the path of the bullet, straight across and then dropping downward to its location. Before attempting to remove the bullet, the fluoroscopic screen was run over the entire body.

To our surprise, a second bullet (Fig. 7) was found in the chest with a very large amount of associated haemothorax.

Point of entry of bullet is not visible on this film but was found to be in approximately the location marked.

A lateral view of the thorax showed the point of entry as indicated by a haemostat clamped to the edge of the wound of entry. The defendant in this case stoutly denied having fired the second bullet, although the

photograph which we saw (Fig. 6) indicated that, after he had fired the bullet into the decedent's head, he could easily have fired a second time while the man was lying in the position as found. In view of the number of bullets that had been fired from various guns by various people during the 'party', it was considered important that both bullets be recovered and that definite evidence be obtained about the principal cause of death;

Fig. 6. (Case 6).

therefore the body was sent to Richmond for complete medico-legal autopsy.

It was established that the first bullet, although passing completely through the brain, had hit no vital centres and that the patient could have lived for several hours after this wound.

The second bullet was determined to be the actual cause of death (Fig. 7). Both recovered bullets were established to have been fired by the same gun. In the courtroom, photographs and X-rays were demonstrated and it is my opinion that the jury probably was more impressed with the X-rays and the extent of injuries by the bullets than with the photographs which were made at the scene of the murder. A conviction of second degree manslaughter with a 5-year sentence was obtained.

Case 7: Wm. M. A Coloured male, aged 65, was admitted to our hospital from a physician in an adjoining county, with a diagnosis of pneumonia.

He died approximately 12 hours after admission with a final diagnosis of pneumonia.

The admission film (an A-P portable X-ray of the chest) showed a

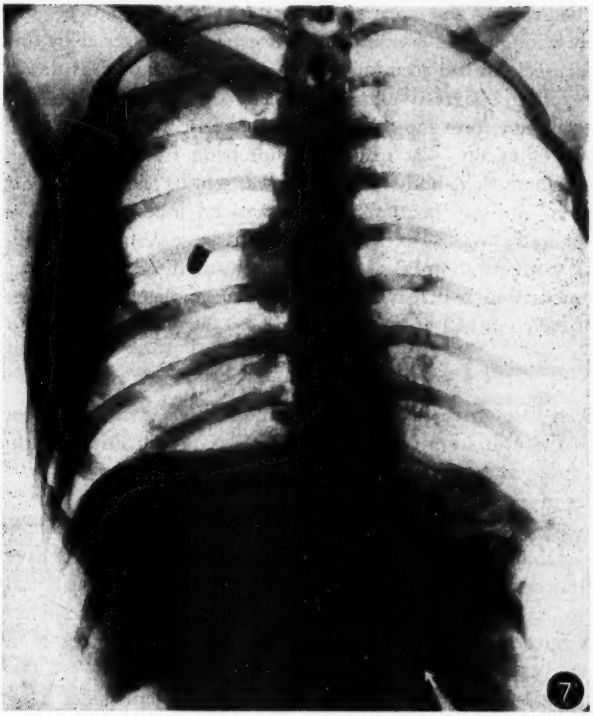

Fig. 7. (Case 6). Second bullet found by fluoroscopic examination. Suspect denied firing this second bullet. This bullet was the actual cause of death. Point of entry noted by marker placed just to left of L2.

pneumonia, but it was not the usual pneumonia. There was emphysema of the left lung and a marked amount of atelectasis in the right lung. This case was referred to the Medical Examiner because of the very short time in the hospital before death. During conversation with the family of the decedent, it was determined that this man had not been well a single day since an industrial injury which he had received 3½ months before. The decedent worked in the log woods for a large industrial concern approximately 60 miles from our hospital. While cutting down a tree, one of the large limbs of a tree struck this patient on the shoulder and knocked him down in a jack-knife position. He was given First Aid and carried to a hospital approximately 70 miles from our hospital where a diagnosis of

3 fractured ribs was extablished by X-ray. The patient was treated for 8 days, then sent home as a convalescent. However, he did not make the expected improvement and began to lose the use of his feet, then his legs and then his hips so that for the last month of his life he had been completely bed-ridden and apparently paralysed in both lower extremities. Strangely enough, this condition had not been discovered by any of the last 4 doctors who had seen him before admission at various times because of his lingering, progressive loss of health.

A lateral film (post mortem) revealed a cause for the development of this chain of events, viz. marked compression fractures of 7th and 8th dorsal vertebrae. The body of decedent was sent to Richmond for complete medico-legal autopsy because of the involved inherent questions which would naturally arise. By this means, it was established that this was an industrial compensation case and the family of the decedent received the full compensation which might have been completely overlooked if the X-ray of the chest had not appeared to be somewhat unusual.

Summary

1. Seven cases have been presented illustrating a variety of conditions in which the utilization of locally available X-ray facilities has proven of definite value as an aid to medico-legal investigation by a rural Medical Examiner in one Tidewater Virginia County.

2. On several occasions, X-ray studies have obviated the necessity for medico-legal autopsy while on other occasions X-ray findings have clearly demonstrated the absolute necessity for complete medico-legal autopsy.

3. Two examples of the use of X-ray records as a means of identification of badly burned human bodies have been shown and a plea is made for further studies in this important type of investigation.

4. Utilization of locally available X-ray facilities provides a rapid and comparatively economical method of determining the cause of death and simultaneously produces a permanent record which may be very effectively used in court room presentation. We believe that in certain conditions, X-ray films may furnish a more effective visuo-educational exhibit than the usual photographs.

5. No expensive or elaborate equipment is necessary. The local hospitals will all have as much as or probably more equipment than has been available in our studies. The primary prerequisites are some degree of scientific curiosity with a high index of suspicion.

I wish, at this time, to express my appreciation to Dr. Geoffrey T. Mann, Chief Medical Examiner, and his staff for their interest, support and co-operation in making this presentation possible. Also, may I express to Dr. Halpern my sincere thanks for his interest and expressed approval of my efforts in this small facet of the complex mechanism of medico-legal investigation.

Possible Neurological Mechanisms Responsible for Sudden Death

With Minimal Anatomical Findings

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The pathologist, functioning as a morbid anatomist, relies upon the autopsy as the time-honoured method to determine the cause of death. He searches for anatomical changes, natural or traumatic, whose presence is incompatible with survival. These lesions he terms the 'anatomical cause of death'. Where anatomical changes are non-specific, meagre or absent, recourse to chemical or physical analytic techniques frequently yields data which clarify obscure problems. There is, however, a group of so-called 'instantaneous physiologic deaths'[1] where anatomical, chemical, physical and micro-biological studies are illuminating only in their negativity. Because such deaths are sudden and unexpected, they come within the purview of the forensic pathologist. The explanation of sudden death in the absence of significant anatomical findings (where toxicological and bacterial studies are negative) emphasizes that the forensic pathologist must function not only as a morbid anatomist but also as a morbid physiologist.

Death which occurs instantaneously without warning symptoms after apparently minor trauma in a healthy child, a robust young adult or a supposed sound middle-aged adult presents a perplexing problem to clinician and pathologist.[2] Clinical diagnoses of fresh coronary thrombosis, cerebral haemorrhage or pulmonary embolism are not corroborated by the pathologist. Only the basic pathological changes of circulatory failure are demonstrable at autopsy, viz. diffuse visceral, mucosal and serosal congestion and capillary haemorrhages. These findings have no specific significance and are present in varying degree in all forms of death. Cessation of vital function stems from cerebral anoxia with no discernible trigger lesion.[3]

Serious disturbance in the function of the circulatory system can arise from mechanical stimuli or stresses which are not necessarily related to,

* Presented at the Fifth Annual Meeting of the American Academy of Forensic Sciences in Chicago, 27 February 1953.

or associated with wound production. Such functional abnormalities are characterized as neurogenic.[4] The methods of examination available to the pathologist or toxicologist to-day are inadequate to disclose the extent or nature of the underlying changes, even though such changes are lethal.[5]

We are thus confronted with the syndrome of *primary shock* or *syncope* with instantaneous exitus. Such unexpected deaths without anamnestic data of pre-existing fatal disease in whom autopsy fails to disclose an organic cause of death constitute a small but real fraction of so-called 'sudden deaths in healthy persons'.[6]

Primary shock and syncope are characterized by suddenly developing circulatory failure on the basis of either reflex vago-cardiac inhibition with slowing or stoppage of the heart, reflex systemic vasodilatation with profound fall in blood pressure, or a combination of these two mechanisms.[4]

Instantaneous death is almost always cardiac in origin.[1, 7, 8] Cardiac standstill occurs simultaneously with collapse and its occurrence depends on an underlying physiological mechanism.[4] Close similarity and interrelationship between the production of instantaneous death and syncope has been noted. Instantaneous death has been considered a form of fatal syncope.[1, 7] Moritz has compared this type of death with the stopping of a clock. At one moment the mechanism is functioning well. A few seconds later all the wheels have stopped. There is an 'abrupt dramatic transition from the glowing activity of life to the lasting stillness of death'.[8]

Inhibition

In 1846 the Weber brothers discovered that excitation of the vagus along its course and stimulation of the peripheral end after section gave rise to marked bradycardia. When excitation was strong enough, complete permanent stoppage of the heart in diastole took place. Subsequent studies have demonstrated that this inhibitory action is not limited to excitation of the vagi but can be accomplished also via a reflex arc.[9]

The term 'death from inhibition' was first used by Brouardel of Paris in the latter half of the last century to describe the clinical entity of rapid and unexpected death due to reflex inhibition of the circulatory and/or respiratory systems. This concept was accepted for a long period, and was eventually abused by being invoked in a wide variety of cases which did not fall within the confines of this syndrome. With continued progress in pathological anatomy, physiology and toxicology the number of deaths attributed to inhibition has diminished progressively.

Kayssi[9] has re-defined deaths from inhibition as those sudden deaths which occur within a few seconds or not longer than 2 minutes after minor trauma or peripheral excitation of relatively simple and innocuous nature. Neither the trauma nor the nervous excitation are *ordinarily* in and of themselves sufficient to cause death directly. No pathological changes able to cause death must be demonstrable at autopsy. There is thus complete

absence of pathological, traumatic or toxic findings. Investigation of the circumstances surrounding death indicates that peripheral irritation or stimulation has probably initiated a fatal inhibitory reflex.

Case: An 8-year-old healthy boy was struck on the chest by a bounding soft ball. He collapsed immediately, vomited once, and was dead within a matter of a minute or two. Complete autopsy disclosed only a few sub-epicardial petechiae. Grossly there was no external or internal evidence of injury or natural disease, and there were no congenital cardiac anomalies. Microscopic examination of all the viscera was non-contributory.

This case is a prototype of death from inhibition. Other varieties of trauma and incidents have been described in the literature, i.e. a blow to the larynx,[10] a punch in the epigastrium, a kick in the scrotum, canalization of the cervix,[11] puncture of the pleura[12] and pressure on the carotid sinus.[13] Post-mortem examination has disclosed either no identifiable organic changes or such minimal alterations as to be readily excluded as the direct cause of death. Impingement of cold water on the nasal and post-nasal mucous membranes after sudden immersion is described as a trigger mechanism for setting off the cardio-inhibitory reflex.[13]

So-called 'pleural shock' is an excellent example of profound and occasionally fatal cardio-vascular collapse on the basis of comparatively minor trauma, i.e. thoracentesis. Autopsies on individuals who died suddenly following needle puncture of the pleura have frequently disclosed no lesion which could be held responsible for death. Air embolism of the arterial variety has been carefully ruled out. Experimental work with dogs has demonstrated that irritation of the pleura, inflamed by either bacterial or chemical means, can lead to rapid and occasionally fatal fall in blood pressure with marked slowing of the heart. The cardio-inhibitory reflex can be prevented or stopped by cutting both vagi in the neck. Atropine destroys the reflex. Irritation of normal pleura does not produce the cardio-vascular response. Inflammation thus sensitizes the sensory end organs and 'primes' the reflex.[12]

Cardiac Syncope

Cardiac syncope may be defined as the sudden loss of consciousness due to cerebral anaemia, which is cardiac in origin. As previously stated, sudden 'cardiac' death is fatal cardiac syncope. Cardiac syncope can be entirely neurogenic in origin.[14]

For the moment I would like to discuss briefly the most common 'anatomical' cause of sudden death, namely, coronary artery disease. Autopsy in many cases of coronary disease (who do not die during a pressor episode) frequently fails to explain sudden death. No organic luminal occlusion by thrombus or intra-mural haemorrhage is present and the myocardium in most cases is capable of maintaining a fairly efficient circulation. Such necropsy evidence indicates that sudden death in coronary disease may be physiological with no organic lesion to explain the onset

of the terminal cardiac dysfunction which leads to sudden circulatory cessation, whether that mechanism be ventricular standstill (asystole) or ventricular fibrillation.[15] There are no anatomical marks by which we can identify a sudden cardiac death of this nature, and the pathologist, no matter how careful his scrutiny may be, cannot, from an examination of the heart alone, say: 'This patient died suddenly. This patient did not die suddenly.'[8]

This situation may thus be regarded as a variety of death from inhibition. Although the individual has chronic vascular disease, there is no apparent reason (eliminating a pressor episode) for sudden cardiac collapse. Similar episodes of fatal (and transient) syncope are seen in cases of aortic stenosis and with Stokes-Adams syndrome.

Anatomical Pathways and Physiological Mechanisms in Death from Inhibition

The mammalian heart is under the nervous control of the autonomic system. Knowledge of cardiac response to autonomic nervous impulses is essential if a physiological basis is to be established for death from inhibition. The sympathetic and parasympathetic nervous systems stand as physiological antagonists, augmenting and inhibiting cardiac function. The adrenergic impulses of the sympathetic system are checked and balanced by the cholinergic impulses of the parasympathetic system.[16]

While the rate of the heart is determined by the pace-maker in the sino-auricular node, the natural tempo is controlled by the balance of antagonistic inhibitory and acceleratory impulses which reach the heart via the vagi (parasympathetic) and the cardiac acceleratory nerves (sympathetic).

The neuro-muscular control of the heart is one of the most sensitive and complicated mechanisms in the body. Brief suspension of myocardial activity leads to fatal cerebral anoxia. It is not so much the absolute degree of alteration in cerebral circulation as the rate of circulatory change that plays the fundamental rôle in the production of fatal and non-fatal syncope.[17]

Stimulation of the right vagus nerve results in equal slowing of the atria and ventricles, decreased vigour of the atrial contractions, increased strength of the ventricular contractions and dilatation of the heart in diastole. Some of the right vagal fibres terminate in the S-A node and decrease its rate of discharge. Stimulation of the left vagus nerve leads to slight atrial slowing with greater slowing of the ventricles and resultant heart block, indicating that fibres from the left side pass to the A-V node.

Strong stimulation of both vagi leads to slowing of the atria with diminution in their contractions and complete heart block with idio-ventricular rhythm and a slow feeble beat. Feeble cardiac contractions result in inadequate coronary blood supply and myocardial anoxia. Asystole or ventricular fibrillation may supervene and death results. Strong vagal impulses begin by depressing the amplitude of atrial contractions. Next they slow the

conduction rate, decrease the ventricular rate and eventually stop the heart entirely.[18]

The autonomic system is constructed on the plan of a reflex arc. The afferent limb of the arc is not necessarily a part of the autonomic system. There is no special group of afferent fibres connected directly with the smooth muscle and glandular cells as there is in the case of the efferent limb. At different times, different fibres may serve as the afferent part of the autonomic reflex arc. *Any* afferent fibre to somatic or visceral regions may carry to the central nervous system impulses capable of mediating a response on the part of smooth muscle, gland or heart. The autonomic system, with its generalized visceral distribution and multiple connexions with the central nervous system, is thus an ideal pre-formed pathway for the transmission of inhibitory impulses, fatal and non-fatal, to the heart.

The vagi contain 4 sets of fibres: somatic and visceral motor, and somatic and visceral sensory. The somatic motor fibres originate in the nucleus ambiguus and the visceral motor fibres originate in the dorsal motor nucleus. Both nuclei are in the medulla. The tractus solitarius carries fibres of somatic and visceral sensation.[19] The motor nuclei are affected directly by higher centres in the hypothalamus and cortex, and reflexly through somatic impulses and vascular impulses arising in the carotid sinus, right atrium and aorta.[18]

While Weiss[1] feels that cardiac syncope is always the result of asystole, Nathanson[15] is of the opinion that asystole is the basis of temporary cardiac syncope and that ventricular fibrillation is the lethal arrhythmia underlying fatal syncope. A combination of cardio-accelerator impulses plus a discharge of epinephrine acting synergistically are capable of throwing the heart into ventricular fibrillation. All *effective* contractions cease and only fibrillary twitchings agitate the surface of the ventricle. Cerebral anoxia supervenes and death ensues.

Many patients who have died during the taking of an electrocardiogram have demonstrated ventricular fibrillation in their records. This arrhythmia may usually be, and perhaps even always is, the last convulsive tremor of a dying heart. Hamman[8] asserts that it is the usual cause of sudden cardiac standstill, since up to this time it is the only satisfactory explanation for sudden cardiac death which has been clearly demonstrated clinically. With acute heart block and complete auriculo-ventricular dissociation, life is temporarily suspended to return again if the heart begins to beat, to depart if it hesitates too long, for then ventricular fibrillation is established. That trivial lesions can inaugurate the fatal rhythm is well known.

Etiological Factors

If the concept of cardiac inhibition is a valid one, why are deaths infrequent which can legitimately be ascribed to it? The events which have been accused of initiating cardiac inhibition are certainly not rare. Blows in the

solar plexus are received in profusion by the professional pugilist, impacts to the scrotal contents are the lot of every active male who indulges in sports, cervical canals without number are irritated every day in gynaecologists' offices, and the professional wrestler has his carotid sinuses stimulated any number of times by his opponent. Apparently inhibition normally passes off sufficiently rapidly, so that functional recovery ensues before myocardial and cerebral ischaemia have gone to the point of irreparable damage. Natural disease of the myocardium is said to render the heart hyper-susceptible to inhibition. Fatty degeneration of the myocardium, fibrosis of the myocardium, myocarditis of various origins and low-grade anoxia due to coronary sclerosis are instrumental in disturbing the cardio-regulatory mechanism to the point where fatal inhibition readily ensues.[4, 11] Alcoholics and those with cerebral disease are cited as being especially prone to this variety of sudden death.[10] Yet many cases of death ascribed to cardiac inhibition demonstrate no acute or chronic lesions.

There is no particular age or sex distribution for death from inhibition. Candidates for this syndrome are said to include the hyperkinetic individual who is sensitive to all types of stimuli, and the individual with an unstable cardio-vascular mechanism who is subject to syncope.[6] The existence of either a constitutional or an acquired state of neuro-circulatory irritability has been postulated.[20] A hyper-active carotid sinus reflex has been described as a possible mechanism.[21] Suffice it to say that at this time there is no way to predict who may fall prey to this catastrophic syndrome. The healthy human mechanism with its adequate emergency reserve can usually re-establish normal equilibrium by the interplay of homeostatic mechanisms. In disease and in some instances where the vital organs according to our present methods of examination are completely normal, return to an efficient level of function is more difficult, slower, or may not occur at all. In the light of our present knowledge we must beg the question of the etiology of death from inhibition.

Clinico-Pathological Correlation

Inasmuch as death in fatal cardiac inhibition results from trauma so minor as to leave minimal or no evidence at autopsy, all possible information concerning the circumstances must be obtained if a solution to the case is to be found. Observations of witnesses and bystanders may offer the only clues for an understanding of what has transpired. The pathologist must leave the dissecting table and continue his search elsewhere if he is to arrive at an answer.

Summary

The essential factor in death from inhibition is the instantaneous establishment of an inhibitory reflex with the sudden arrest of a vital organ by what has been termed 'micro-trauma',[9] injury of so minor a degree as to leave

no or minimal visible evidence at autopsy. There are no characteristic post-mortem appearances or findings.

When available, details of the circumstances of death usually describe some definite precipitating event.

Death from inhibition may be diagnosed only by *exclusion*, with fatal organic disease eliminated by a complete autopsy and a thorough toxicological analysis.

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Estimation of Stature from the Long Bones

A Discussion of its Reliability

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The biometrical relationship between the length of the various long bones and the stature of the individual has been extensively quoted and used by medico-legal authorities, and almost all text-books give instruction in calculating the stature from isolated bones. The authorities quoted are, as a rule, either Rollet, Manouvrier, or Pearson. Many authors have pointed out the theoretical and practical difficulties in using Manouvrier's tables or Pearson's formulae on populations other than the particular population from which the tables and formulae were derived. These difficulties may briefly be summarized as follows:

1. Both Manouvrier's tables and Pearson's formulae were derived from figures published by Rollet[6] (1888), who measured the cadaveric length and the 'fresh' long bones (i.e. with articular cartilage intact) of 50 male and 50 female subjects in Lyons in the south of France. The mean age of these subjects was about 60 years, and the mean stature rather low, about 5 ft. 5 in. for the males, and about 5 ft. for the females.

Manouvrier[4] (1892) eliminated subjects over 60, and was left with 24 males and 25 females. He made no other adjustment for the effect of age on stature. His tables, from which can be read cadaveric length from the length of the 'fresh' long bones, contain 20 readings for each sex. It is evident that many of his readings, especially at the extremes of the tables, were based on isolated observations.

Pearson[5] (1899) used all Rollet's material, but made no allowance for ageing. His great contribution was the introduction of regression equations as a form of analysis of the figures; his mathematical method has not been improved.

2. Pearson's formulae have been found by several observers to underestimate the stature of other European populations. Pearson (1899) himself had this experience when he estimated the stature of 7 executed French criminals and compared the results with their known statures. The mean estimate was 2·73 cm. (more than an inch) too low. Trotter and Gleser[10] (1952) were able to do the same on 100 American White male subjects, and

found that the mean estimate based on the length of the femur was 3·67 cm. (about 1½ in.) too low; when the humerus was used the error rose to 6·22 cm. (about 2½ in.). Manouvrier's tables are even less satisfactory in this respect than Pearson's formulae, at any rate as regards the femur.

3. The various racial groups differ sufficiently in their body proportions to make tables or formulae derived from one racial group unsatisfactory for use on another population. Stevenson[7] (1929) first showed this when he attempted to use Pearson's formulae on Chinese material, and 2 recent American studies comparing American White and Negro groups have clearly shown the necessity of using figures derived from the population under investigation. The errors involved are particularly relevant in a country like South Africa, where no series of observations on the local population groups have yet been published. These errors will be further considered below.

Three recent publications and one critical survey indicate a re-awakening of interest in the subject. Telkkä[8] (1950) published measurements of 115 male and 39 female Finnish subjects, and included a fairly complete bibliography in his paper. Dupertuis and Hadden[2] (1951) analysed figures from 100 male and 100 female American White dissecting room subjects, and similar numbers of male and female American Negro cadavers. Comparing their mean cadaveric statures with means derived from several large series of living Americans, they concluded that their cadaver length was equivalent to living stature.

In December 1952 there appeared an outstanding contribution from Mildred Trotter and Goldine C. Gleser[10], of Washington University, St. Louis. For the first time they were able to compare the lengths of long bones with the *known living stature* of American military subjects, all of whom had been measured in a reasonably similar way when they joined the Army. For their principal White male group they were able to study 710 subjects in this way. A parallel study on dissecting room material enabled them to observe directly the change of stature between living and post-mortem measurements. They were thus able to show that Dupertuis and Hadden's assumption was incorrect, the average increase in stature produced by the change from living to corpse measurements being 2·5 cm. They also tested Dupertuis and Hadden's formulae on the bones of 100 American military White male subjects, and found that these formulae seriously overestimated the known living statures of the subjects. Trotter and Gleser also used a correction for ageing derived from a painstaking partial correlation study made by the same authors[9] (1951) on a total of 855 dissecting room subjects. The correction was a loss of 0·06 cm. per year after 30 years of age, and they showed close agreement between this figure and one derived from Rollet's original figures. Trotter and Gleser's paper is by far the most thorough yet published, and deserves close study by those who may be required to give evidence about stature estimation.

Modern Trends in Forensic Medicine, edited by Keith Simpson, was published in 1953, and includes a survey of the subject by Prof. J. D. Boyd and Dr. J. C. Trevor. Unfortunately they did not have access to Trotter and Gleser's recent paper,[10] and therefore relied heavily on Dupertuis and Hadden. They follow these authors in stating: '. . . it now seems probable that, whatever the reason may be, a greater correspondence exists between recumbent corpse length and upright living stature than has been supposed'. It has already been pointed out that this is likely to be incorrect. They also recommend the use of Dupertuis and Hadden's 'general' formulae, which these authors derived from a weighted average of Pearson's and their own White and Negro results for each sex. Trotter and Gleser have criticized these formulae, and state: 'A "general" equation or an average of the equations derived from different racial groups would necessarily result in poorer estimates of stature for any particular group or individual to which it is applied than would an equation derived from a similar group. . . . And, if the stature of a single individual from such a mixed group were desired, the equation most likely to give the most accurate estimate is that pertaining to the race and sex most frequently represented in the group.' The apparent success of Dupertuis and Hadden's 'general' formulae is partly due to the fact that their own over-estimation is to some extent corrected by Pearson's under-estimation.

The error in using regression formulae for reconstruction of stature from long bones is statistically expressed as the 'standard error of the estimate'. This is the measurement of the error likely to be made in reconstructing the stature of an individual known to belong to a population similar to that from which the regression formula was calculated. (The additional error involved when the formulae are used on individuals from other racial groups will be considered separately below). The use of the standard error of the estimate (s.e.e.) will be made clear by an example. The s.e.e. of Pearson's formula for an estimate of stature from the length of the femur is 3·2 cm. Suppose the best estimate of the stature is 180 cm. The difference between the true stature and this estimate may be expected to exceed the limits of:

± 3·2 cm., i.e. 176·8—183·2 cm. in 1 case out of 3;
± 6·4 cm., i.e. 173·6—186·4 cm. in 1 case out of 22;
± 8·0 cm., i.e. 172·0—188·0 cm. in 1 case out of 100;
± 9·6 cm., i.e. 170·4—189·6 cm. in 3 cases out of 1,000.

(The second, third and fourth lines express the chances of exceeding the limits of ± 2, 2·5, and 3 times the s.e.e. respectively). It is clearly not safe to rely on the odds of 2:1 expressed in the first line, and it is common statistical practice to regard any deviation of less than twice the standard error as being due to chance. In other words, the estimate made in this hypothetical case cannot be expressed in terms more precise than: 'The individual from whom the femur was derived is unlikely to have been taller than 186·4 cm. (6 ft. 1½ in.) or shorter than 173·6 cm. (5 ft. 8½ in.).' No opinion

could be offered on the exclusion of any individual whose stature fell within this range of 5 inches.

The published standard errors for the use of formulae based on the femur, and on the humerus, are shown in Table I.

TABLE I: STANDARD ERROR OF THE ESTIMATED STATURE IN CM.

	Femur		*Humerus*	
	Male	*Female*	*Male*	*Female*
Pearson (1899)[5]	3·2	3·2	3·2	3·4
Telkkä (1950)[6]	4·9	4·0	5·0	3·9
Dupertuis and Hadden (1951)[2]				
Whites	3·4	3·3	4·6	4·1
Negroes	4·3	3·6	5·1	4·7
Trotter and Gleser (1952)[10]				
Whites	3·3	3·7	4·0	4·5
Negroes	3·9	3·4	4·4	4·3

The smallest standard errors of the estimate published are 3·0 cm., recorded by Trotter and Gleser[10] for the estimation of the stature of male American Whites from the lengths of tibia and femur combined; and one of 2·3 cm. from a multiple regression equation of Dupertuis and Hadden on male American Whites involving the lengths of femur, tibia, humerus and radius. Trotter and Gleser (1952)[10] state that the figures from which this last formula was derived do not show a sufficient range of stature to provide fully satisfactory equations.

Errors naturally increase when the formulae are used on subjects from different population groups. In addition the results will be biased in the direction caused by the differences in body proportions between the groups. Stevenson[7] (1929) found that Pearson's formulae underestimated the stature of his Chinese subjects by 4·5 cm. on the average. More recent comparisons between the two American groups, Whites and Negroes, are found in the two American studies already quoted. Trotter and Gleser[10] show that in their subjects the Negroes 'have longer limb bones relative to their stature than do Whites', in addition to the well known greater length of the forearms and the legs relative to the arms and the thighs. Therefore when they calculated the mean stature of their Negro males using their White male formula for the femur, they overestimated it by 4·3 cm.; the corresponding figure from Dupertuis and Hadden's study was 2·6 cm. The bias in the opposite direction, i.e. from calculating the mean stature of their White male group using their Negro male formula for the femur, was 3·8 and 3·6 cm. respectively. Such a bias significantly increases the standard error of the estimate, though naturally the bulk of the errors occur in the direction indicated by the bias.

In the review by Boyd and Trevor[1] already quoted the only guide to the errors which may be expected runs as follows: 'Owing to the inconsis-

tencies mentioned above no very definite indication can be given of the error which may be made in estimating the stature of a particular individual. For the formulae involving the length of a single bone it is probably seldom less than 2·5 cm. (1 inch), but those involving the lengths of two or more bones can be expected to give somewhat more reliable figures.' It is felt that this is misleading; while the error will often be less than 1 inch, it cannot be *relied on* to be less than 2½ inches, even in the most favourable circumstances. An estimate differing from a missing man's stature by ½ inch therefore provides no greater proof of identity than one 2½ inches different.

It will be clear that in South Africa, where no figures for local populations are available, all one can do is to use Trotter and Gleser's two sets of formulae, bearing the chances of greater error and possible bias firmly in mind when drawing up reports and giving evidence.

Fragmentary Remains

A fairly celebrated South African case, referred to as the Wolkersdorfer case, was reported by Gordon and Drennan[3] in 1948. It necessitated identification of burnt and fragmentary remains. Only the proximal ends of a humerus and a femur were recovered, and estimates had to be made of the length of the whole bones before an estimate of stature could be attempted. For the humerus it was stated 'that the distance between the head and the lower limit of the deltoid impression usually amounts to about 52% of the total length of the bone', and for the femur 'that the length from the top of the greater trochanter to the nutrient foramen, although variable, usually amounts to about 34% of the total length of the bone'. For neither bone were any details given of the variability of the proportions alluded to.

On a subsequent occasion I was consulted about the application of the method in a similar case. Following the recommended procedure a femur of 508 mm. and a humerus of 313 mm. were estimated from the stumps available. By any set of formulae these lengths resulted in estimates of stature differing by over 6 inches. There was no doubt that the 2 fragments derived from the same cadaver; the result stimulated me to re-examine the theoretical basis of the methods described.

I first examined 100 random femora. Three of these had no nutrient foramen, and one had a foramen near the distal end of the bone. Of the remaining 96, 25 had 2 nutrient foramina and one had 3. Taking the proximal foramen in those bones which had more than one foramen, the mean proportion as described by Gordon and Drennan was 35·1%, with a standard deviation of 4·2%. A simple calculation shows that even if the estimation of stature from the length of a femur were completely reliable, the uncertainty introduced by the variability of the proportion involved a standard error of the estimate of stature of about 10 cm. Doubling this

produced a range of ± 8 in., and would clearly be useless for any identification purposes, including as it would almost any adult person. It follows that no reliance can be placed on the position of a nutrient foramen in reconstructing a femur from a proximal fragment.

In the case of 100 humeri, measured in the recommended way, the mean proportion was 52·3% with a standard deviation of 2·1%. Calculating again on Trotter and Gleser's formula for the humerus,[10] this variability itself involved a s.e.e. of about 3·7 cm., which combined with the s.e.e. of 4·0 cm. when the length of the humerus is known, makes a s.e.e. of 5·5 cm. (2·2 in.). Twice this amounts to 4¼ in. While it cannot be maintained that this figure is too high to be of any use at all, it is clear that an estimate reading, for example, 'between 5 ft. 2 in. and 5 ft. 10½ in.' will be of limited value in narrowing down the possibilities.

It follows that in the Wolkersdorfer case the close agreement between the statures estimated from the fragmentary femur and humerus was fortuitous, and the agreement of each with the known height of the deceased equally so. The identification of the deceased was not materially influenced by the estimation of his probable stature, though it was naturally regarded as confirmatory evidence.

Summary

The present practice in the estimation of stature from long bones is examined, and the objections to the use of the usually quoted authorities outlined. The recent literature is reviewed.

The errors involved in these estimations are examined in detail, and the proper form in which the results should be stated is explained.

The methods used in the Wolkersdorfer case are examined critically.

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Estimation of Age from Cranial Suture Closure

A Report on its Unreliability

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The degree of closure of the various sutures of the vault of the adult skull is often used in determining its age for medico-legal purposes. The alleged estimation of an 'exact' age period is more often the rule rather than the exception. It is the purpose of this paper to indicate that such assessments on an individual adult skull cannot be made with any degree of accuracy because both endocranial and exocranial closure in any given suture vary very widely.

Detailed observations have been made on 100 Cape Coloureds, 190 Bantu, 50 White Germans, 60 North American Indians and 30 Eskimos; but in this short report examples are chosen of known age and sex which will show degrees of sutural obliteration unexpectedly different from those described in the standard text-books. These specimens are in no way abnormal; they are merely normal variants of which there are many other examples.

A suture develops when 2 adjacent bones of the skull come into close relation. It is relatively wide in infancy and later forms a narrow, immovable joint between the 2 bones. The connective tissue spanning the sutural 'gap' is a remnant of the membranous capsule of the brain connecting the outer and inner periosteum of the 2 bones and is usually termed the *sutural ligament*. This sutural connective tissue is gradually reduced in thickness, while the simple straight sutural line may also undergo extensive complications by the formation of interdigitating ossified processes of the 2 bones. However, adjacent edges of a suture may be flat and linear, bevelled in varying but constant directions, or overlapping.

The fact that the sutures undergo obliteration with age was first pointed out by Vesalius and clearly demonstrated by his pupil Fallopius (Montagu[6]). The state of sutural closure has been used to evaluate the age of the skull ever since the sixteenth century.

During the last hundred years a number of papers has been published concerning this matter. Some conflicting conclusions have been arrived

at, but one of the interesting facts is that as early as 1885 Topinard did not have a high opinion of the value of suture closure as an indication of age, but he only had small series of skulls to base his views on. Similar opinions were held by Frédéric[3] and Zanolli.[13] Dwight[2] stated (1890):

'It must not be forgotten that there are other guides (than suture closure) to the age of the skull; and I am not prepared to assert that taken together with them, the sutures are absolutely worthless in the hands of an experienced anatomist. I am sure that to anyone else the rules in question are misleading and dangerous.'

Hrdlicka[4] considered that 'of all the marks that the adult stage of life has been reached, the most handy and reliable is the occlusion of the basilar (basisphenoid) suture'. The usual age given for this is 25 years, though I have seen individuals of 23 years with a fused suture and of 27 years with a partially open suture. Hrdlicka also pointed out that in estimating the age of a skull, the obliteration of the cranial sutures is a feature which is 'useful when taken conjointly with other characters'. Furthermore, he maintained that with experience and taking the condition of the sutures and teeth together, one might estimate the age of an adult subject perhaps to within 10 years. The present author is convinced, however, that (in the adult) the state of the surface wear of the teeth is a hazardous index of age, especially in the many ethnic population groups in Southern Africa with their varying diets, customs and environment. It may be stated that the present method of age estimation of adult skulls found in Africa is quite unsatisfactory.

In 1924 Todd and Lyon[8] started to publish a series of papers dealing with endocranial and exocranial suture closure. Thus far these constitute one of the major contributions to the problem of the ageing of the skull. Their figures are most widely quoted and form the basis of quite dogmatic statements in the various text-books on forensic medicine. Sometimes the reference source is not clear in such a text-book, but on further investigation the figures can be traced back to Todd and Lyon or earlier investigators. For example, in *Medical Jurisprudence*,* by W. F. Rhodes, I. Gordon and R. Turner, the chapter on *Identity* is written by M. R. Drennan and J. A. Keen. Their quoted authority (mentioned in their footnote to Fig. 9) is Drennan's report in *Arquivos do Instituto Benjamin Baptista* (Rio de Janeiro, **5**, 27, November 1939). This investigation (which was to estimate the duration of life in the Bushman as determined by the suture closure of his skull) was based partly on the work of Todd and Lyon and partly on that of Martin. Martin's results were, in turn, based on earlier workers' data obtained from observations on small groups of European skulls where ages were often not known and unsatisfactorily gauged from tooth wear. Drennan mapped out a scheme for the approximate ages of closure of various sutural segments of a Bushman's skull based almost entirely on his own estimation of the sequence of closure of sutures in 53 Bushman

* Published by The Stewart Printing Co., Pty., Ltd., Cape Town. 1945, 2nd ed.

skulls whose age was in the first place *estimated* to within 10 years (i.e. a statistical range of 21 years) by Martin's figures[5] (1928) (obtained from European skulls) and then modified by Todd and Lyon's figures (alleged to be accurate to within 6 years, i.e. a span of variability of 13 years taking an actual figure as midpoint). Thus, although the data and diagram presented by Drennan and Keen are taken from Drennan's paper in the *Arquivos do Instituto Benjamin Baptista*, the actual information they present concerns Europeans (as determined by Martin, and Todd and Lyon), the Bushman data not being included. This type of information, which may readily be quoted in any court of law, is misleading, but this is not an isolated example. Even in Simpson's *Modern Trends in Forensic Medicine**, J. D. Boyd and J. C. Trevor, discussing the estimation of age at death in human remains (pp. 142-143), quote Todd and Lyon's data without reservation or criticism, and only urge caution by referring to Dwight's comments (quoted above).

Todd and Lyon awarded numerical values to signify varying degrees of suture closure, thus:

0 = 'open' suture not fused at any point;
1 = commencing union;
2 = half obliterated suture;
3 = more than half obliterated; and
4 = completely obliterated suture.

The process of obliteration is brought about by small tongues or 'bars' of ossified tissue stretching across the sutural gap, these 'bars' linking up slowly along the length of a suture, as seen from the inside or outside, until union is complete. This method allows easy manipulation of a mass of data and was adopted by the present author, who only examined the vault sutures as these are the most useful for observing age changes.

Each of these sutures is subdivided as follows:

The *coronal suture* into 4, numbered 1 to 4, No. 1 being nearest the midline landmark, the bregma, where the sagittal suture meets the coronal (Fig. 1). Todd and Lyon only had 3 subdivisions here, so that the present writer's second and third portions together equal their part No. 2.

The *sagittal suture* is also subdivided into 4 parts (numbered 1 to 4, from before backwards so that No. 1 is nearest bregma).

The *lambdoid suture* is subdivided into 3 parts (numbered 1 to 3, No. 1 being nearest midline point, lambda, where the sagittal meets the lambdoid suture).

The sagittal is a single suture, whereas the other 2 are duplicated on the right and left side of the skull. Consequently the state of closure of each suture may be represented by a formula, e.g. sagittal 0.3.4.1, indicating that the first portion of the sagittal suture (nearest the bregma) shows no union; the second portion is more than half obliterated; the third portion is completely united; while the fourth portion shows commencing union.

For the purpose of comparison, the conclusions of Todd and Lyon, relevant to this paper, will be summarized:

1. Suture closure in general becomes apparent upon both exocranial and endocranial

* Published by Butterworth & Co., Ltd., London, 1953.

surfaces of the skull almost simultaneously. Exocranial closure progresses more slowly shows more individual variation. It is hence less reliable as an age indicator.

2. The greatest activity in closure, both exocranially and endocranially, occurs between the years 26 and 30. Periodicity is further manifest in secondary periods for activity in the later thirties (exocranial), the fifties (both) and the late seventies (endocranial).

3. The sutures of the vault commence closure first, in the following order:

In the sagittal at the age of 22 years, the coronal at 24 years, and the lambdoid at 26 years of age, and move rapidly to near complete closure at about 30, after which progress to completion is slow, at 35, 41 and 47 years, respectively.

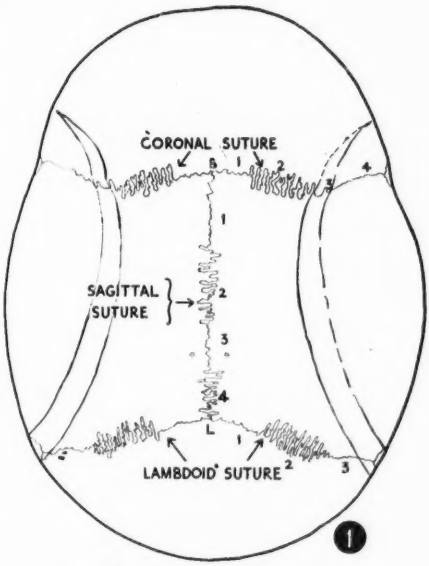

Fig. 1. Diagrammatic representation of the subdivisions of the cranial vault sutures.
B = Bregma.
L = Lambda.
Sagittal: 1 = pars bregmatica.
2 = pars verticis.
3 = pars obelica.
4 = pars lambdica.
Coronal: 1 = pars bregmatica.
2 = pars complicata.
3 = pars stephanica.
4 = pars pterica.
Lambdoid: 1 = pars lambdica.
2 = pars intermedia.
3 = pars asterica.

The ages of commencement and termination of closure in the vault sutures, both exocranial and endocranial, in White males and American Negroes are summarized in Table 1.

It must be emphasized that their conclusions and most symmetrical graphs cannot be relied upon entirely, as (to mention merely one instance) only male crania were examined and of these, those showing 'irregularities of closure' were automatically eliminated. They also felt that eliminations for irregularities in closure would have reduced prohibitively the already inadequate female series, so these were completely discarded. Various reasons were also given for 'smoothing out' the graph curves, but these excuses cannot really be considered valid. Other features of handling and interpreting their material are also open to severe criticism. It therefore seems difficult to understand how so much reliance has been placed, since 1924, on their 'selected' data.

In any case, even accepting a fair degree of accuracy in the data chosen,

the data in Table 2 indicate the extreme variability of suture closure both exocranially and endocranially. These skulls have been chosen to show that, *in any individual skull*, the age of the individual at death cannot be estimated from the degree of closure of the various cranial sutures, whether taken individually or collectively or whether observed exocranially or endocranially. The data may be compared with those in Table 1 for either the White or the Negro, for there is apparently no 'racial' difference in closure patterns.

TABLE 1: AGES OF CRANIAL SUTURE CLOSURE (according to Todd and Lyon, [8-12], modified from Cobb[1])

	Exocranial				*Endocranial*			
Suture	*Male White*		*Male Negro*		*Male White*		*Male Negro*	
	C	T	C	T	C	T	C	T
Sagittal	20	29	20	32	22	35	22	31
Coronal 1 & 2	26	29	23	32	24	38	24	38
Coronal 3	28	50	25	35	26	41	25	44
Lambdoid 1 & 2	26	30	23	31	26	42	23	46
Lambdoid 3	26	?30	22	?31	26	47	27	46

C = Commencement; T = Termination.

Conclusions

In this short report a detailed analysis of the above information is not required. The following conclusions emerge:

(*a*) In the skull aged 25 years, the sutures are well advanced towards obliteration, when, by convention, they should hardly have commenced;

(*b*) In the skull aged 27 years according to Todd and Lyon, all 3 sutures should already have commenced uniting and should be about half closed;

(*c*) The skull aged 30 years, on the other hand, shows scarcely any closure; and

(*d*) The remaining 7 skulls show marked differences from the closure times of Todd and Lyon (Table 1), the closure pattern being in conformity with what would have been expected previously at a much younger age (Figs. 2A-D).

The total closure units are given above so as to gauge more easily the degree of closure of the suture as a whole. Although it may appear, in these few examples, that endocranial closure is far more advanced than the exocranial, in a large survey these differences may 'iron out' in a graph, but this matter is beyond the scope of the present report.

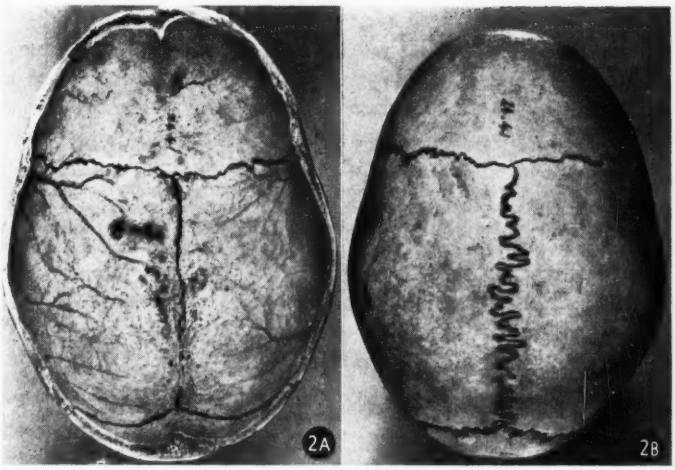

Fig. 2A. Endocranial Aspect: There are a few 'bars' of ossified tissue stretching across the sagittal suture in the obelion region (subdivision 3). Note that the remaining sutures are wide open. In general, it may be said that the closure of the endocranial aspect of the sutures is almost as sporadic and whimsical as that of the exocranial aspect.
Fig. 2B. Exocranial Aspect: The sutures show a remarkable degree of patency for an individual in the fourth decade of life. A small 'bar' of union is seen towards the lower end of the sagittal suture (pars obelica) opposite a large *parietal foramen.*

Fig. 2C. Exocranial Aspect: Here again, the exocranial view belies the *expected* age, though, endocranially, the coronal and lambdoid sutures (Table 2) are in agreement with what was 'expected' of a person between 50 and 60 years.
Fig. 2D. Exocranial Aspect: For an individual of more than 60 years, with nothing clinically to explain the obvious patency of the sutures, this skull is even more remarkable than that shown in Fig. 2B, because the endocranial aspect (Table 2) is less advanced in degree of closure.

TABLE 2: THE DETAILED ANALYSIS OF SUTURE CLOSURE IN SELECTED SKULLS OF KNOWN AGE

Specimen No.	Stock	Age	Sex	Coronal						Sagittal				Lambdoid					
				Exocranial		Total Closure Units Possible: 32	Endocranial		Total Closure Units Possible: 32	Exocranial	Total Closure Units Possible: 16	Endocranial	Total Closure Units Possible: 16	Exocranial		Total Closure Units Possible: 24	Endocranial		Total Closure Units Possible: 24
				Left	Right		Left	Right						Left	Right		Left	Right	
272·466	WG	24	M	4314	4314	24	4444	4444	32	4443	15	4444	16	110	110	4	331	331	14
57·46	CC	27	M	0001	0001	2	0000	0000	0	0000	0	0000	0	000	000	0	000	000	0
41·46	CC	29	M	1104	2114	14	3334	3444	28	1333	10	3344	14	311	311	10	341	341	14
46·45	CC	30	F	0101	0112	6	0000	0000	0	0020	2	0000	0	000	010	1	010	000	1
51·45	CC	35	M	1011	1110	6	1000	3110	6	1000	1	3111	6	000	000	0	100	100	2
29·43	CC	41	F	0010	0010	2	0010	0000	1	0000	0	0100	1	000	000	0	000	000	0
24·41	CC	42	F	0010	0010	2	0002	0000	2	0010	1	1130	5	000	000	0	000	000	0
33·44	CC	43	M	0001	0001	2	0000	0000	0	0130	4	0030	3	100	100	2	000	000	0
272·470	WG	54	M	1114	1004	12	4444	4444	32	3343	13	4444	16	110	220	6	332	330	14
6·47	CC	58	F	0011	0030	5	4444	4444	32	0010	1	2312	8	000	000	0	341	340	15
73·42	CC	64	F	0000	0104	5	0000	0424	10	0000	0	0000	0	000	000	0	000	000	0

CC = Cape Coloured; WG = White German.

Summary

Evidence is provided that, with techniques available at present, an assessment regarding the precise age at death of any individual, gauged only on the degree of closure of the vault sutures of the skull, is a hazardous and unreliable procedure.

I am thankful to Drs. T. Dale Stewart and Marshall Newman of the National Museum, Smithsonian Institution, Washington, D.C., and to Prof. Raymond Dart, University of the Witwatersrand Medical School, for permission to make detailed surveys of their anthropological material. I am grateful for research grants from the University of Cape Town Staff Research Fund and the Dr. C. L. Herman Research Fund, and for a Fellowship from the International Rotary Foundation which enabled me to carry out research in the United States of America.

I also wish to thank Mr. D. J. Coetzee for the sketch and Mr. G. McManus for the photographs.

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REVIEWS OF BOOKS

MODERN TRENDS IN FORENSIC MEDICINE

Modern Trends in Forensic Medicine. Edited by Keith Simpson, M.D. (Pp. 327 + ix, with 133 illustrations. £3 7s. 9d.). London and Durban: Butterworth & Co. Limited. 1953.

Contents: 1. Forensic Aspects of Stillbirth and Neonatal Death. 2. Pathology of the Lung in Stillbirth and Neonatal Death. 3. The Investigation of Sudden Death. 4. Recent Work on Changes After Death. 5. Forensic Aspects of Blood Groups. 6. Exposure to Cold—Starvation and Neglect. 7. Problems in Reconstruction. 8. A Review of Physical Aids in Criminal Science. 9. Electro-Encephalography in Forensic Medicine. 10. Modern Trends in the Civil Law in Relation to Medical Practice. 11. Recent Advances in Toxicological Analysis. 12. Toxicology of the Newer Insecticides and Selective Weed-killers. Index.

For many years there has been a serious gap in our knowledge of recent advances in medico-legal investigation and research. We have been entirely dependent upon the new editions of standard text-books which obviously can only appear at infrequent intervals. For this important reason, *Modern Trends in Forensic Medicine* is to be welcomed as an overdue contribution to contemporary forensic teaching and practice. The Editor has assembled a distinguished list of expert contributors and he has himself been responsible for several extremely practical chapters.

An important topic discussed by Dr. Osborn is the pathology of the lungs in stillbirth and neonatal death. This chapter is profusely and excellently illustrated and reviews much basic research done by the author himself. It should be read in conjunction with Dr. Keith Simpson's own admirable account of the forensic aspects of this problem. Dr. Osborn's statement, however, that a rise in the carbon dioxide content of the foetal blood results in 'dark fluid blood' (p. 35) is in conflict with the more acceptable statement by Dr. Mant who (in Chapter 4) reviews Mole's important investigations and concludes that 'uncoagulable fluid blood is normally present in . . . any healthy person who dies a sudden natural or unnatural death from almost any cause. Fluid blood at autopsy suggests merely a sudden death, certainly not necessarily one from asphyxia' (p. 96).

The position of the highly controversial subject of status lymphaticus and its bearing upon sudden death for trivial reasons is also reviewed in the light of modern knowledge of the adrenal glands. The emphasis, however, is to be laid on the adrenal cortex and not on chromaffin tissue, as suggested on p. 60.

In an interesting chapter dealing with the investigation of sudden death Dr. Keith Simpson draws attention to the extremely low incidence of coronary disease among male Negroes in the United States. A valuable contribution to this problem has been made by Brink from Johannesburg who has worked out the anatomical pattern of the coronary tree in the Bantu heart (*Clinical Proceedings*, 1949, **8**, 137).

Dr. Simpson's volume also includes important new work on the chemical composition of the cerebrospinal fluid and the way in which the time of death (in the first 15 hours after death) may be calculated with much greater precision than is possible on the basis of the information provided by rigor mortis and the temperature of the dead body.

This important work was initiated by Schourup in Denmark and certainly needs confirmation and extension. In 50 cases Schourup was able to calculate the exact time of death in 34%. He was able to establish an exactness of $\pm$ one hour in 84% and an exactness of $\pm$ 1½ hours in all 50 cases. The method is only applicable in persons over 15 years of age and injuries must not have allowed the escape of cerebrospinal fluid. If the method is established it appears to offer 'a 3-hour margin up to 15 hours after death with an 80% probability of the time falling within the middle 2 hours' (p. 84).

The review of data for the establishment of identity and the related problems of race, sex, age and stature, especially as based on skeletal remains, provides one of the most up-to-date contemporary surveys of this important field. Here is an extensive analysis of anthropometric data which emphasizes the importance of allowing for such factors as sex differences in the determination of age and generally makes available to the forensic expert a digest of the extensive anatomical

researches which have been conducted most assiduously in recent years.

It should, however, be pointed out that in estimating age at death in human remains, the external measurements are not reliable for the estimation of conceptional age in prenatal human material. There are normal variations in length for a specific age, and the amount of shrinkage that occurs is difficult to assess.

Too little emphasis is placed on the variability of the appearance of ossification centres from the viewpoints of individual variation, sex, race and environmental factors (e.g. nutrition).

Although Boyd and Trevor suggest that great care should be exercised in age assessment from suture closure, a disproportionate amount of data on the age of closure based on the work of Todd and Lyon is presented. Their data should come under review because of its highly selective nature. Only male skulls were examined, and then only those without 'irregularities of closure'. These irregularities are the very variations of the normal which need to be taken into account.

Dr. Hill's paper on brain waves brings to general notice the way in which electrical discharges from the brain may be of use in our Courts. The interpretation of these tracings is obviously in the sphere of the specialist in this field and it is interesting to find that, although abnormalities have been found in association, e.g. with a psychopathic personality, schizophrenia and epilepsy, 'the limited conclusions which may be reached from the study of the criminal by electroencephalography should not be stretched or distorted in an endeavour to accommodate them to the forensic needs of the individual case. The contribution which electroencephalography might aspire to make is not therefore to assist the prisoner through the narrow McNaghten gate, but to throw some light upon the physiological constitution which is of relevance to the study of his behaviour and his adaptive capacity within a physical and psychological environment.

Compared with the very large number of established data relevant to the social and psychological factors in crime, the contribution of the EEG is indeed meagre. What can be stated? Certain groups of criminals have mild abnormalities in the EEG about 3 times as commonly as the general population, unselected. These abnormalities are found even more frequently in patients suffering from frank mental disorders. There appears to be a relationship between such EEG patterns and aggressive behaviour disorders and the possessors of such EEG's are probably more than normally susceptible to physiological and psychological imbalance when exposed to chemical stresses.

Nothing is known about their susceptibility to psychological stresses and this is an inviting but would be a very difficult field of research. Since the incidence of EEG abnormality is directly related to age, declining as age advances, and the type of pattern seen in the adult may be normal for the much younger subject, it is assumed that the phenomena reflect an immaturity of cerebral organization. This is further supported by the observation that the behaviour of the abnormal adult frequently bears resemblance to the behaviour of the normal but frustrated child'.

Dr. Hill emphasizes that the McNaghten Rules are based upon a theory of psychiatry now long abandoned. The Rules require that, in order to be free from blame, 'the accused did not know what he was doing, or if he did know, he did not know that it was wrong'. As Dr. Hill points out (pp. 233—234) 'only the *unconscious* subject does not *know* what he is doing, and there are very few states of changed consciousness which leave the subject capable of co-ordinated purposeful action. Probably only in the dazed state following an epileptic discharge, involving during the discharge complete abolition of consciousness, is this possible. Moreover, a man may *know* perfectly well what he is doing at the time he performs an act, but subsequently have a genuine amnesia for the act. This undoubtedly occurs sometimes in states of alcoholic excitement'.

In a subsequent edition of this invaluable contribution to contemporary forensic literature, there may well be room for a discussion of the concept of responsibility and, indeed, of the more novel and recent Scottish concept of diminished responsibility associated with certain psychopathic abnormalities. The lawyer and the psychiatrist are usually completely at loggerheads in this field and a necessary preliminary advance in our understanding of crime and the nature of its punishment requires a frank discussion based on what modern scientific knowledge can provide.

Both the Editor and the publishers are to be congratulated upon the production of this admirable, distinguished and timely work.

THE ACID BATH MURDER

The Trial of John George Haigh. (*The Acid Bath Murder*). Edited by Lord Dunboyne. (Pp. 271 with illustrations. 15s.) London: Edinburgh: Glasgow: William Hodge and Company, Limited. 1953.

Contents: Introduction. Chronological Table. *The Trial:* 1. First Day—Monday, 18th July, 1949. Evidence for the Prosecution. 2. Second Day—Tuesday, 19th July, 1949. Evidence for the Defence. Appendices.

The trial of John George Haigh has come to be known as the *Acid Bath Murder* and by this macabre description it has captured the imagination of lay as well as professional readers.

In an admirable introduction Lord Dunboyne (who has edited this *Trial* most admirably) summarizes the facts of the case very succinctly (p. 19): 'All the ingredients of a horrifying mystery were being unearthed, and, to the popular mind, the case was a *cause célèbre* of the first order. In its essentials, the capital charge, formulated against Haigh, was not difficult to appreciate: a financially embarrassed man of apparent charm had ingratiated himself with a widow of some wealth, inveigled her into a death trap, dissolved her body in sulphuric acid and reported her to the police as a missing person. Then, when cornered, the accused heaped horror upon horror, fully describing the crime without any trace of remorse and coolly claiming to have killed eight other human beings and to have drunk their blood'.

The Defence adopted an unusually bold and imaginative course. There was almost no cross-examination of the Crown witnesses except for a few questions to about four witnesses calculated to throw light on the abnormal mental condition of the accused. In addition, the only witness called for the Defence was an eminent psychiatrist who set out to establish that Haigh suffered from a paranoid constitution (i.e. anomaly of personality) which had developed into that rare but highly characteristic disorder, viz. paranoia. On this hypothesis Haigh's murders were divinely appointed and it was also under a divine impulse that he is alleged to have drunk his own urine, an act which (on a specific occasion) he demonstrated he was capable of doing, but which he had not otherwise been seen to do while under observation.

Dr. Yellowlees, who provided the psychiatric assistance for the Defence, was apparently completely convinced of Haigh's paranoia; but the problem remained that Haigh apparently committed his murder at a time when he was hard-pressed for money, that he made detailed preparations for the act well in advance of the killing and that he took every possible precaution to ensure that he would not be discovered. It is difficult to relate such careful scheming to the statement elicited from Dr. Yellowlees under the acid cross-examination of Sir Hartley Shawcross (the Attorney-General) that Haigh is alleged to have told him (Dr. Yellowlees), in connexion with the murder of another victim, that the urge came upon him as a result of his dreams and that 'he (Haigh) could not have told a minute before the event that he was going to kill anybody' (p. 180).

Dr. Yellowlees was obviously on extremely difficult ground, and the general problem is discussed particularly well in an interesting Appendix contributed to this edition of the *Trial* by Dr. Clifford Allen on the *Medical Aspects of the Case*. Dr. Allen was certainly not prepared to go further than that 'Haigh certainly suffered from a psychopathic personality and, less evidently, from a sadistic element. He murdered for profit but was able to do so because he was an abnormal type, being neither entirely sane nor entirely insane.

Such cases are not necessarily insane according to the McNaghten Rules, although whether the Law is completely satisfactory is a matter for further discussion' (pp. 270—271).

The relentless cross-examination of expert witnesses by skilful and highly trained legal minds is also well brought out in this *Trial*. Experts cannot escape their previous testimony in other cases and the following passage (p. 182) shows into what a difficult corner conscientious witnesses can be pushed:

'You have given evidence in a great many cases where the defence has been insanity?—Yes, I have indeed.

And you have not previously had difficulty, have you, in saying whether or not you thought the prisoner concerned knew or did not know that what he was doing was wrong?—It depends on what you mean by difficulty.

You have given evidence upon the point, at all events?—Yes.

This is the first case, is it not, in which you have been unable to say whether or not in your view the prisoner knew that what he was doing was wrong?—Yes, I think that is true.'

This case abounds in many intriguing medical and legal points. Particularly astute was the demonstration by means of a precipitin test that gall stones found in the sludge which remained after the decomposition of the victim's body by sulphuric acid were in fact of human origin. This, together with the damning evidence of the acrylic dentures which resisted acid decomposition, provided valuable proof of the identity of the deceased. (It should be mentioned in passing that, if the routine laboratory precipitin tests were used, these do not in fact establish that the material tested was human. It is not possible, from the scientific point of view, to establish any more precise a category than that of the primates).

It will also be recalled that this *Trial* was remarkable for the action taken against the *Daily Mirror* which had referred to the accused after his arrest and before his case had been opened as a vampire, thus prejudicing the accused before his case was heard. The Editor of the newspaper was committed

to prison for a term of 3 calendar months and the newspaper company was fined £10,000 and ordered to pay the costs of the proceedings.

This is one of the most interesting and valuable additions to the *Notable British Trials* series and forms a useful complementary volume to the *Trials* of Peter Griffiths, Neville Heath and Ronald True. In all these cases skilful but unsuccessful psychiatric lines of defence were explored.

HALSBURY'S LAWS OF ENGLAND

Halsbury's Laws of England. Editor-in Chief: The Rt. Hon. Lord Simonds. Advisory Panel: The Rt. Hon. Lord Goddard, Lord Chief Justice of England; The Rt. Hon. Sir Raymond Evershed, Master of the Rolls; The Rt. Hon. Lord Merriman, President of the Probate, Divorce and Admiralty Division. Editor: J. T. Edgerley. Durban: Butterworth and Company (Africa) Limited.

The third edition of this important legal work has been announced by Butterworth and Company (Africa) Limited and the first volume is already available. The scope of the undertaking will comprise a complete statement of all matters of substantive law.

An Annual Cumulative Supplement will be issued, incorporating all recent developments, and arranged under the titles of the main volumes, so that the latest position can be determined by a single reference to the current Supplement.

Apart from the individual Index, Tables of Cases and Tables of Statutes for each volume, there will be a consolidated Table of Cases and a consolidated Index for the whole work, to be produced as soon as possible after the completion of the text volumes.

The new edition will consist of 42 volumes and the volumes will be published approximately quarterly.

SIMPSON'S FORENSIC MEDICINE

Forensic Medicine. By Keith Simpson, M.D. (Lond.). (Pp. 344 + viii, with 131 figures. Second edition, 21s.). London: Edward Arnold and Company, 1952.

Contents: I. *Forensic Medicine.* 1. Signs of Death. 2. Changes After Death. 3. Identification. 4. Blood-Stains. 5. Types of Injuries and Wounds. 6. Fire-Arm Wounds. 7. Asphyxia. 8. Regional Injury. 9. The Ultimate Effects of Injury. 10. Natural Disease and Trauma. 11. Infanticide. 12. Abortion. 13. Sexual Offences. 14. Legal Procedure. 15. The Medico-Legal Autopsy and Evidence. 16. Medical Ethics. 17. Medico-Legal Aspects of Insanity. II. *Toxicology.* 18. Laws Regulating Sale of Poisons. 19. General Facts about Poisons. 20. Corrosive Poisons. 21. Irritant Poisons. 22. Hypnotic and Narcotic Poisons. 23. Deliriant Poisons. 24. Convulsant Poisons. 25. Paralytic Poisons. 26. Abortifacient Drugs. 27. Gaseous and Volatile Poisons. 28. Gaseous and Volatile Poisons. 29. Poisoning by Plants, Flora and Fungi. Index.

Dr. Keith Simpson (Reader in Forensic Medicine to the University of London) has been responsible for this essentially practical manual, the popular usefulness of which is well attested by the fact that a second edition became necessary within the space of five years.

The new edition has provided him with opportunities to incorporate additions made since the first edition appeared in 1947 and to make extensive changes in the illustrations.

It is always difficult to know how much information should be imparted to the undergraduate student. Forensic medicine may not loom large in the medical curriculum from the academic point of view, but the medical practitioner is caught up as an expert in most intricate and difficult problems concerning which he may have to assist the Court as an expert witness on most crucial issues. The subject, therefore, probably merits more serious consideration in the planning of the medical student's studies. It is probably also desirable that more undergraduate time should be devoted to the medico-legal instruction, even though the modern syllabus is already a very full one.

Dr. Keith Simpson's book is admirable in that, in a convenient compass, it provides an adequate introduction to the subject. The author's need to condense the presentation of his material unavoidably leads to a certain amount of dogmatism and this frame of mind is undesirable in forensic practice. Although he states, e.g. that the distinction between ante-mortem and post-mortem wounds presents no great difficulty, (p. 15) there are undoubtedly medico-legal pathologists who would not agree with this proposition. His cautious qualification of this statement, especially in connexion with recently dead bodies before rigor mortis has developed fully and before the body has cooled considerably, is to be commended.

Dr. Keith Simpson also draws attention to the fact that the blood may remain fluid after death for some time and that, indeed, it may never clot at all. The significant implications of this important observation are not fully appreciated by many forensic experts. The fluidity of the blood *post mortem* is often relied upon as one of the signs that death was due to asphyxia. Modern investigation has demonstrated that this is an entirely unwarranted assumption and that fluid blood in the cadaver is entirely non-specific and without diagnostic meaning. This point was well brought out and applied by Dr. Camps in the Setty case (reported in the *Medico-Legal Journal*, 1951, **19**, 2). Contemporary knowledge certainly requires a revision of the statement on p. 86 that the blood is

likely to remain fluid longer in cases of asphyxia because of the greater degree of cyanosis.

The thorny problem of status lymphaticus and its relation to sudden death for trivial causes is very ably reviewed (pp. 139—140). Selye's epoch-making researches on the adrenal cortex and stress undoubtedly require a fresh evaluation of this problem in the light of modern endocrinology, especially as status lymphaticus has an important bearing upon the interpretation of the cause of certain types of anaesthetic and other unexpectedly sudden deaths.

Dr. Keith Simpson's book also contains a valuable section on toxicology. It will continue to fulfil a useful and important rôle in undergraduate teaching.

PSYCHOLOGICAL DISORDER AND CRIME

Psychological Disorder and Crime. By W. Lindesay Neustatter, M.D., B.Sc., M.R.C.P. *With a Foreword by* John Maude, *Q.C.*, Recorder of Plymouth. (Pp. 248. 21s.). London: Christopher Johnson.

Contents: 1. Some Popular Fallacies. 2. 'Guilty but Insane'. 3. The Common Treatments of Psychiatry. 4. The Psychoneuroses. 5. Schizophrenia. 6. Depression and Mania. 7. Loss of Memory and Fugue States. 8. Psychopaths and Irresistible Impulse. 9. Mental Deficiency. 10. The Organic Psychoses. 11. The Electroencephalogram and Epilepsy. 12. Head Injury. 13. Alcoholism and Carbon Monoxide Poisoning. 14. The Sexual Perversions. 15. Murder. 16. Theft. 17. Juvenile Delinquency. 18. Treatment or Punishment? Appendix. Bibliography. Index.

Although this book is intended for the non-medical (especially the legal) reader, the author has performed his task so well that it will undoubtedly be of interest to the medical practitioner in general practice (because of the excellent manner in which this account of psychological disorders is given). Moreover, because the subject is viewed throughout from the forensic standpoint, it forms a useful guide to those (especially beginners) who are required to give expert testimony in our courts.

The discussion of the irresistible impulse is extremely instructive. Dr. Neustatter makes the important point that the compulsive neuroses are the very ones that usually do not result in crime. If we exclude psychotic behaviour, we may well ask whether there is such a thing (in medical language) as an irresistible impulse. Indeed, is not the impulse really one that has not been resisted? (p. 96).

The author also makes the homely point that the so-called truth drugs, like gin, merely loosen the tongue and that patients can and do tell lies very effectively while under the influence of these drugs. They 'neither ensure the revelation of the truth nor prevent the patient from lying' (p. 83).

Dr. Neustatter contributes an interesting review of sexual disorders and is very optimistic about the use of female sex hormones in the treatment of certain types of pervert. He also draws attention to the civilized way in which sexual offenders are handled in the Psychopathic Institution in Denmark. This institution is a hospital rather than a prison, and offers the basis of a practical solution for the treatment and possible rehabilitation of the psychopathic offender in general.

The volume will undoubtedly be of great benefit to the legal practitioner who must brief himself for handling problems in this field. The expert witness will also save himself many a discomfiture by studying carefully the experienced advice which this volume offers. It will certainly help those involved in this work to tread warily when they give evidence in the courts.

MEDICO-LEGAL NOTES AND NEWS

THE TWELFTH ITALIAN CONFERENCE OF LEGAL AND INSURANCE MEDICINE

This Congress will be held in Bari from 8—11 October 1953. Prof. G. Bianchini of Siena University will report on the *Medico-Legal Problems of Tumours*.

Those wishing to communicate papers are invited to send the title, together with a short summary, to the Secretariat of the Congress without delay.

Further information may be obtained from Prof. A. Franchini, Direttore dell'Istituto di Medicina Legale e delle Assicurazioni dell' Universita di Bari (Pallazzo dell 'Ateneo).

AMERICAN ACADEMY OF FORENSIC SCIENCES

The Fifth Annual meeting of the American Academy of Forensic Sciences was held at the Drake Hotel in Chicago from 26—28 February 1953.

Over 60 papers were presented at a very successful meeting.

BRITISH ASSOCIATION OF FORENSIC PATHOLOGISTS

The next meeting of this Association will be held in November in England this year. Those interested in obtaining further information should communicate with Dr. Francis Camps, 79 Harley Street, London, W.1.